DISEASES OF RASPBERRIES

and

Erect and Trailing Blackberries

Agriculture Handbook No. 310

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CONTENTS

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DISEASES OF RASPBERRIES and Erect and Trailing Blackberries

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INTRODUCTION

The first part of this handbook concerns the diseases of raspberries.[2] Following this is a section on the diseases of erect and trailing blackberries.[3] In each of the two sections virus, fungus, and bacterial diseases are discussed, in like order. The diseases within a section are presented in alphabetical order of their common names. For major diseases, and to the extent there is sufficient information for others, diseases are discussed under the following topics: General symptoms, causal organism (or virus), disease cycle, host resistance, and control. Finally considered are some diseases caused by nematodes, and some noninfectious ones common to all cultivated *Rubus* species.

Throughout the publication wild or unusual *Rubus* hosts are identified in accordance with current botanical terminology as well as by common name. Where possible, the terminology used is that found in the Index of Plant Diseases (Agriculture Handbook No. 165, U.S. Department of Agriculture (*305*)[4]). Authorities are not in general agreement about species limits in the genus *Rubus* (*121*, *137*), and references to some species are presented as given by the authors cited.

Terminology of the virus, fungus, and bacterial diseases follows the usage of the Index of Plant Diseases (*305*) except where recent literature indicates a change.

The monograph by Kennedy, Day, and Eastop (*194*) is followed for insect nomenclature except where current usage in the United States is at variance with it.

[1] The assistance of W. R. Jarvis, J. D. Menzies, and R. Stace-Smith in criticizing the manuscript is gratefully acknowledged.

[2] "Raspberries," as used in this publication, refers to raspberry plants and their fruits in the genus *Rubus*, subgenus *Idaeobatus*. Unless specified otherwise the term concerns the species and the derivatives of *Rubus strigosus* Michx. (=*R. idaeus* L. var. *strigosus* (Michx.) Maxim.), American red raspberry, and the species and the derivatives of *R. occidentalis* L., black raspberry. The hybrids between these species, sometimes termed "× *R. neglectus* Peck," purple raspberries, are included.

[3] "Blackberries," as used in this publication, refers to plants and fruits of the erect and trailing (dewberry) species in the subgenus *Eubatus* of the genus *Rubus*. The erect species are represented typically by *Rubus allegheniensis* Porter, Allegheny blackberry; *R. argutus* Link (=*R. ostryifolius* Rydb.), highbush blackberry; and *R. frondosus* Bigel., yankee blackberry. "Trailing blackberries" refers to plants and fruits of the prostrate species in the subgenus *Eubatus*, in which a large number of *Rubus* species and species hybrids are involved in the genetic composition of various cultivars (*137*).

[4] Italic numbers in parentheses refer to Literature Cited, p. 88.

While the diseases important to cultivated *Rubus* in the United States receive primary consideration in this handbook, reference is made to the European literature on these diseases, and some exotic diseases of possible importance in the United States are also considered. Many viruses, fungi, and bacteria cause diseases in raspberries and blackberries in the United States that seriously damage the crops. Some diseases occur generally on many of the cutivated species of *Rubus*, while others are limited to a few types of brambles or to certain regions of the United States.

The most effective control measures for raspberry and blackberry diseases are those taken before the diseases become serious. The decisions made when choosing planting stocks and planting sites are usually the most important in determining freedom from disease problems, especially virus diseases. Horticulturally desirable varieties that are adapted to a locality and are resistant to the major diseases occurring there should be planted, if available. Many of the fungi and all of the bacteria and viruses causing raspberry and blackberry diseases can be brought in on diseased planting stocks. Stocks certified as being substantially disease free by a State plant inspection service should be used where possible. Satisfactory soil, water, drainage, and cultivation and management practices that promote vigorous growth are also very important in growing healthy *Rubus* crops.

A recent, excellent review of the bramble viruses (*276*) mentions 11 virus diseases of cultivated brambles that are known to occur in the United States; these and some additional virus diseases will be treated in this publication.

In the United States there are 84 different species of fungi, distributed among 67 different genera, and 4 species of bacteria, in 2 genera, recorded as occurring on cultivated brambles exclusive of ornamentals (*305*). Recognized diseases are caused by 53 of these fungi; 24 are minor pathogens or secondary invaders of dead or dying host tissue and 7 are necrophytes on dead canes. Among the bramble diseases of fungal or bacterial origin of measurable economic importance somewhere in the United States, 35 will be considered in this handbook in some detail. About 50 other diseases will be noted in passing. Most of these diseases of brambles in the United States were discussed (1956) in the excellent text by Anderson (*6*); and in 1958 the literature on *Rubus* diseases, particularly as regards host resistance, was abstracted (*199*). Two British texts (*47*, *341*) also contain useful descriptions of *Rubus* diseases.

There are several disorders that either have not been associated with any pathogen or are known to arise from the effects of the physical environment on the plant. Certain of these important noninfectious disorders will be discussed in this handbook.

DISEASES OF RASPBERRIES

Virus Diseases

Raspberries probably are more seriously damaged by virus diseases than any other fruit crop in the United States. Once infected, the plants do not recover. All vegetative parts of a virus-infected plant

are commonly infected so that suckers or tips taken from such infected plants will also be diseased. Some raspberry viruses are carried by certain aphids that can spread them to healthy raspberries in the same field. No known raspberry viruses are spread in the field by pruning or otherwise mechanically injuring the plants under natural conditions.

Most viruses infecting raspberries and the aphids known to be the vectors of these viruses are limited largely to *Rubus* hosts. Some of the viruses are seed borne (*207*, *217*) and some are also known or thought to be transmitted by ectoparasitic nematodes (*39*, *57*). The host range and mode of spread of some viruses infecting raspberries are still largely unknown.

Virus diseases of *Rubus* have recently been ably reviewed by Stace-Smith (*276*), Cadman (*57*), and Smith (*269*). *Rubus* viruses in Czechoslovakia were reviewed by Helebrant (*157*), in Estonia by Tiits (*302*), in Finland by Tapio (*296*), in Norway by Bjørnstad (*31*), in Yugoslavia by Jordović (*191*), and in Europe in general by Baumann (*17*).

General measures for control of virus diseases of raspberry consist of (1) selecting healthy stock; (2) using resistant varieties where possible; (3) removing virus-infected *Rubus* as far as practicable from both cultivated and wild healthy stock; (4) frequently inspecting and roguing the plantings; and (5) controlling aphid vectors in raspberry fields.

Black Raspberry Streak

The virus diseases severe streak and mild streak are recognized as separate entities by some authors (*84*, *183*, *266*), but as not separate by others (*276*). Experimental transmissions of severe streak by grafting, by vector, or mechanically are not reported in the literature. The workers who have dealt with mild streak have felt that these two diseases are distinct, and do not intergrade. Lending plausibility to this view is the fact that the virus nature of mild streak has been established. Although the relationship between the two diseases has not been determined, they are separable on the basis of symptom severity on black raspberry (*84*, *247*). They are described separately here.

Severe streak.—Severe streak is found on black and purple raspberries. There is a possibility that severe streak occurs on blackberry with or without symptoms (*320*). It is lethal to black raspberries, killing the plants in a few seasons. The disease is not widespread, however, occurring mostly in scattered areas in Ohio and western New York. The disease was first described in 1923 (*320*). The assumption that it is caused by a virus is still based primarily on the inability to find other etiological agents.

Symptoms.—The leaf symptoms of severe streak are first apparent soon after flowering. The tip leaves of young canes show a peculiar curling (fig. 1, *A*). The tip of the leaf is at first sharply hooked or recurved, and later is curled downward and backward, sometimes being rolled into a cylinder. If a young plant has severe streak when set in the field, by August or September the tips of the upper leaves are recurved or curled, and by fall the plant is weak and sickly. Bushes infected after they are planted produce hooked leaves in a

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FIGURE 1.—Severe streak of black raspberry leaves: *A*, Infected leaves, the midribs hooked or recurved; *B*, plant in the rosette stage; *C*, normal plant.

few weeks. In either case, the next year the leaves of new shoots and laterals will be hooked, and the fruit will be small and inferior. The margins of leaflets are not curled downward as for raspberry leaf curl, nor are the veins depressed. Curling is most severe on the younger leaves of rapidly growing plants.

By midsummer, the leaves of chronically infected plants, especially those of the fruiting canes, show a definite mottling, the yellow and green areas not being so well defined as in mosaic on black raspberry. The green areas are not blistered and elevated. The leaves of plants that have been infected for a year or more are close together, giving the appearance of a rosette in extreme cases (fig. 1, *B*); the canes of such plants are shortened.

The characteristic from which the name streak is obtained is the irregular discoloration of the young canes (fig. 2). If the infection occurs early in the season, the canes will usually show a few dark blue dots or irregular streaks before the wood ripens that fall. If the plants were infected the previous year, the symptoms appear on the young shoots near the ground in June or July. At times the cane discoloration does not appear. The discolored streaks affect only the outer green layers of the canes, and do not extend into the wood or pith. Some symptoms of Verticillium wilt may be confused with severe streak. Broad and continuous streaks of blue or black should be noted as usually accompanying infection by the *Verticillium* fungus (see Verticillium Wilt, p. 46); these Verticillium wilt streaks are deep, and they extend from the ground up one side of the cane, sometimes even to the tip. Frequently, the canes with severe streak disease do not survive the second winter after infection; but when infected plants live, their canes may root at the tips for a few years until the disease becomes severe.

In neglected plantings, the symptoms of severe streak are less conspicuous than in well-cultivated fields of vigorous plants (*320*). The symptoms develop best in hot weather, and their intensity can be controlled experimentally by regulating the greenhouse temperature.

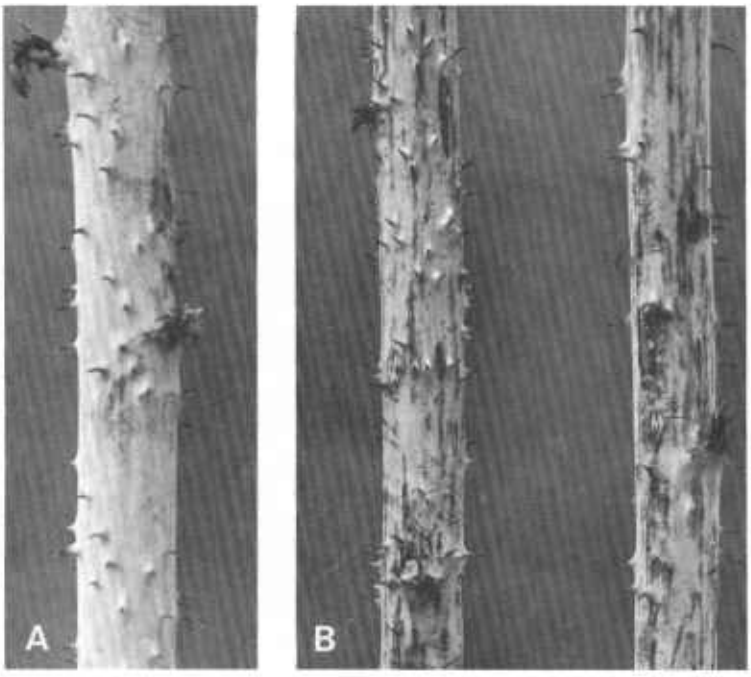

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FIGURE 2.—Severe streak of black raspberry canes: *A*, Normal cane; *B*, canes showing severe streak symptoms.

Disease cycle.—The means of spread of severe streak is unknown. However, there are some reports that black raspberries develop streak faster in the vicinity of wild blackberries (*299*) or cultivated blackberries (*85*) than when isolated from them. The virus assumed to cause severe streak is not soil retained (*320*) and is not transmitted by *Amphorophora rubi* (Kltb.), *A. sensoriata* Mason, or *Aphis rubicola* Oestl. (=*A. rubiphila*) (*22*). Aphids or leafhoppers have frequently been suggested as vectors but without supporting data.

Control.—Severe streak is best controlled by use of clean planting stock, prompt roguing of suspected plants, and possibly by removal of wild black raspberry and blackberry plants from the fence rows surrounding the raspberry planting.

Mild streak.—Mild streak is restricted, so far as is known, to black raspberry, and possibly to blackberry (*84*). In black raspberry it is sometimes a serious problem, particularly because of the difficulty of detection. Mild streak occurs in the Northeastern United States and has been reported as widespread in Maryland, western New York, and western Pennsylvania. The early literature on mild streak has been reviewed (*168*, *183*).

Symptoms.—Symptoms are similar to those of severe streak, but are less obvious. The severity of symptoms may vary from season to season among varieties of black raspberries and on a given plant. Discolored streaks, often rather faint, develop on the lower portions of the young canes during the summer (fig. 3, *A*). The streaks are usually

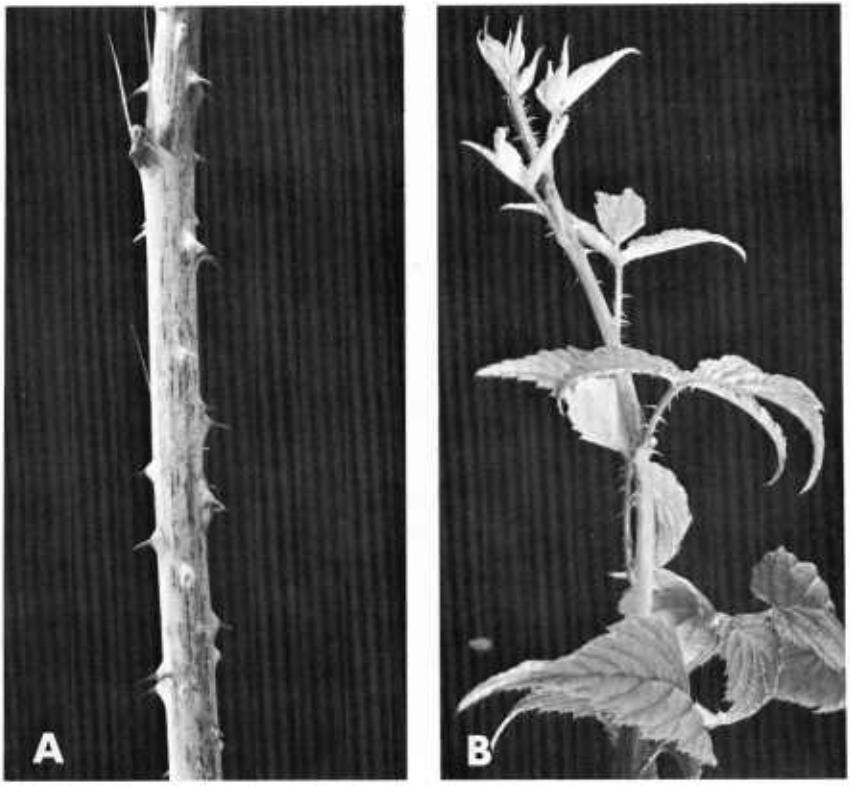

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FIGURE 3.—Mild streak of black raspberry: *A*, Cane showing numerous faint streaks; *B*, shoot showing cane streaking and hooking of leaves.

numerous, purplish, water-soaked lines, each less than 1 inch long. Midribs of leaves on new canes are often hooked and recurved, causing the leaves to be twisted (fig. 3, *B*); and vein-clearing or mottling may develop in lower leaves. Plants are generally vigorous and propagate well. Black raspberry fruits of infected plants are only three-fourths normal size (*99*) and are seedy, insipid, dull, crumbly, and of very poor quality (*84*) (fig. 4). The drupelets of a given fruit often ripen unevenly (*84*).

A fruit disorder known as "brown berry" was considered by Zundel (*360, 361*) to be part of the symptomatology of mild streak on black raspberry. However, Demaree (*99*) pointed out that brown shriveled berries may occur in the presence or absence of mild streaks on canes, and he considered brown berry to be a separate disorder of unknown etiology (see fig. 16, *B*).

The virus.—The virus nature of mild streak was established by successful approach-graft transmission from infected to healthy black raspberries in 35 of 45 trials and by successful dodder transmission (*Cuscuta subinclusa* D.&H.) in 8 of 30 trials (*168*). *Fragaria vesca* L. and some commercial strawberry varieties have been reported to

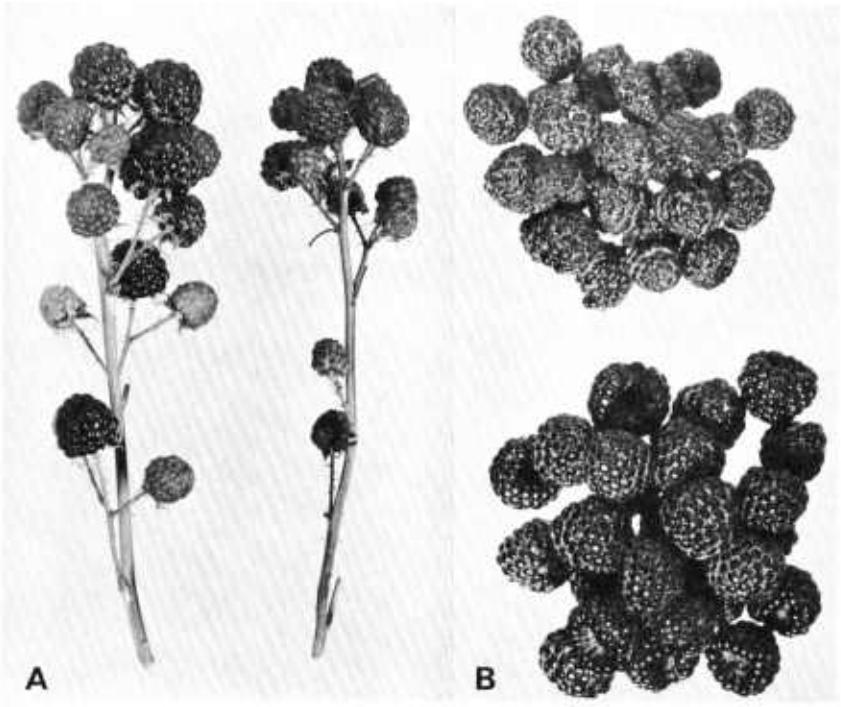

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FIGURE 4.—Mild streak of black raspberry fruit: *A*, Left, healthy; right, infected. *B*, Upper, infected; lower, healthy. Note the larger size and higher gloss of the healthy berries.

develop symptoms when leaf-grafted with mild-streak-infected black raspberry (*38*). Mechanical transmission from black raspberry to black raspberry was unsuccessful (*168*).

Disease cycle.—Mild streak is readily spread in nursery stock because it is difficult for nurserymen and growers to detect. The natural means of spread are not known; but *Aphis rubicola* Oestl. (=*A. rubiphila*) (*247*), *Amphorophora rubi* (Kltb.) (*247*), *Amphorophora sensoriata* Mason (*168*), *Deltocephalus flavicosta* Stal. (*168*), and *Philaenus leucophthalmus* L. (*168*) from infected field plants failed to transmit the virus. Mild streak sometimes spreads rapidly in the field (*183*), and spread is more rapid near existing infection foci than it is at a distance from such foci (*247*, *339*).

Host resistance.—No resistant varieties of black raspberry are known except Black Beauty (*339*) and Improved Kansas (*84*), both reported to remain free of the disease in infected fields.

Control.—The principal control of mild streak is avoidance of stock infected with the virus. Prompt roguing of infected plants is beneficial. The incidence of mild streak in black raspberry increased in the vicinity of wild *Rubus* (*183*), which should therefore be removed near the planting. Proximity to cultivated blackberry is said to increase mild streak (*182*).

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FIGURE 5.—Leaf curl of red raspberry.

Leaf Curl

Raspberry leaf curl is an important and easily recognized virus disease found on red, black, and purple raspberries. It occurs most frequently in the Rocky Mountain region and from Minnesota east into the New England States. There are two strains of leaf curl virus that attack red raspberries. One strain (alpha) attacks only red and purple raspberries. The other strain (beta) attacks red, purple, and black raspberries (principally the black), but produces more severe symptoms on red raspberry than the alpha strain.

Symptoms on red raspberry.—The first symptom of leaf curl is usually manifested on the tips of vigorously growing newly infected canes. The leaflets in the tip regions appear rounded with their margins curled downward. This condition does not spread during the same season to other canes already growing in the hill, but all canes growing later will show curl symptoms. The following spring, both the old canes and the young shoots will have their leaves conspicuously curled and dwarfed (fig. 5), and the young shoots will be shorter than normal. Sometimes shoot proliferation develops. Each year the plant becomes less vigorous; and each year the new shoots are shorter, until finally they are only a few inches in height. After a cane shows curled leaves, its fruit is valueless for market purposes (fig. 6). Ripening prematurely, the fruit is small, dry, seedy, and unpleasant to the taste

When diseased shoots first show above the ground, they are of a pale, yellowish-green color. During the summer, the spaces between the veins on the leaf often remain light green, but later they may become

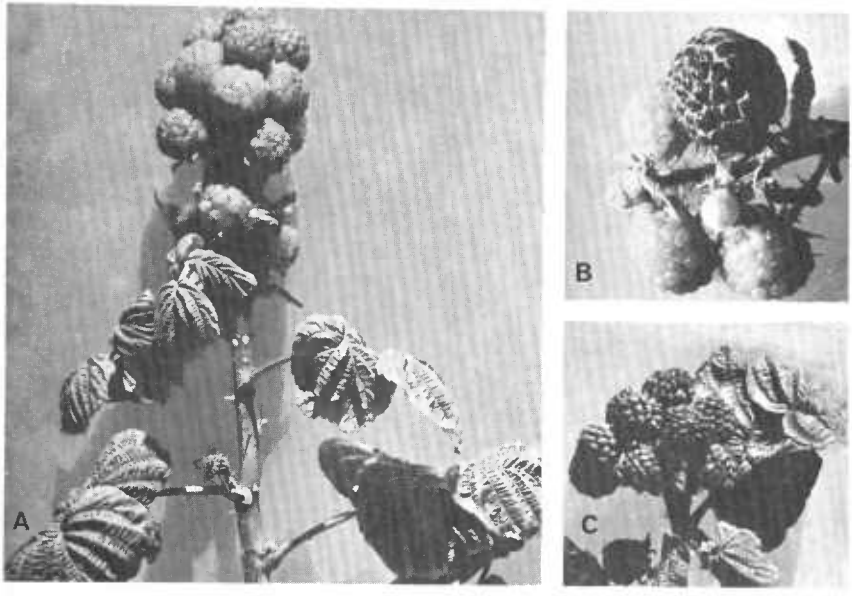

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FIGURE 6.—Leaf curl on fruiting canes of black raspberry: *A*, Dwarfed leaves with sunken veins; *B*, fruit from a healthy cane; *C*, shriveled fruit on a diseased cane.

reddish brown. This is most noticeable on shooots that appear in late summer.

Symptoms on black raspberry.—The beta curl virus infecting black raspberry produces similar symptoms to those produced on red raspberry by the alpha strain, which will not infect black raspberry (*23*, *77*). In a comparison on red raspberry, the beta strain produces more severe symptoms than the alpha. On black raspberry the most conspicuous symptoms of leaf curl are found on the tip leaves, which are arched upward as if by contraction of the margins. In general, veins of infected leaves are sunken, with interveinal tissue ridged or arched upward. Veins are usually darker in color than the tissues between. Leaves become firm and rigid in appearance and remain small and circular in outline. The symptoms often appear first on a single cane. The following year, all canes of a plant are affected. The plant is bushy, and much dwarfed, and its berries are small, dry, and worthless. Young canes are stiff and brittle, and frequently do not branch. New canes are shorter each year until finally the plant dies. During the first season following infection, the canes occasionally root at the tips. In later stages, they are short and stiff and will not bend to the ground to root at the tips.

The viruses.—The raspberry viruses that are known as curl or leaf curl viruses are quite distinct from the Scottish raspberry leaf curl virus (*55*) (see table 1, p. 19), but they were rather recently incorrectly classed with this virus (*257*). The raspberry viruses, differing in host range but causing similar symptoms, were described by Bennett (*23*). Alpha-curl virus infects red and purple but not black raspberry, while beta-curl virus infects black as well as purple raspberry and infects red raspberry somewhat more severely than the alpha-curl virus (*23*, *77*). The alpha-curl virus is heat stable in the host (*279*).

Disease cycle.—A small, sluggish raspberry aphid (*Aphis rubicola* Oestl.) is the vector of both of the raspberry leaf curl viruses (*23*). Winged forms of the aphid that develop in June may spread the virus to healthy stock over distances of a few miles from infected cultivated or wild plants (*23*). The aphids may acquire alpha-curl virus in 2 hours of feeding on infected plants and remain viruliferous thereafter (*22*). Transovarial transmission of alpha-curl virus was not found (*267*). *Aphis rubifolii* (Thos.), common on blackberry, and *A. spiraecola* Patch, occasional on red raspberry, did not serve as vectors of beta-curl virus (*77*); but the European aphid *A. idaei* V.d.G. was a vector of alpha-curl virus in British Columbia, where the aphid was recently discovered (*279*).

Host resistance.—The two curl viruses occur in wild red and black raspberries and in wild wineberry (*Rubus phoenicolasius* Maxim.). Blackberries, on which the disease is rare and unimportant, will harbor the viruses (*22*), in some cases in symptomless condition (*77*).

No widely grown varieties of red raspberry are at present curl resistant. The black raspberry variety Plum Farmer is immune from curl (*77*, *266*), and the New Logan variety is said to be resistant in some areas (*265*, *266*) but not in Michigan. Other commonly grown black raspberry varieties are susceptible, as are the purple raspberry varieties.

Control.—Use of clean stock, isolation from diseased fields, roguing, insect control, and use of resistant varieties—all measures discussed in more detail for raspberry mosaic control (p. 17) are applicable for raspberry leaf curl control. Roguing can be done anytime during the growing season, since symptoms that once appear are never masked. Symptoms from new infections may be slow to appear, however. The same procedures are used for curl vector control as for raspberry mosaic vector control (see pp. 17–18).

Mosaic

Viruses comprising the raspberry mosaic complex are the most widespread, and probably cause the largest economic losses of any raspberry viruses in the United States. Symptoms vary with the variety of raspberry infected, the viruses involved in the mosaic complex, and the season of the year. Mosaic is most readily seen in the cooler weather that occurs in late spring and again in the fall. Leaf symptoms are masked by hot summer weather. Black raspberries are the most severely injured by raspberry mosaic; no raspberries are known to be immune. Strains of the two component viruses of the raspberry mosaic complex are sometimes symptomless, and usually are not as damaging to the host singly as when they occur in combination (*275*). The range of symptoms of raspberry mosaic described here is caused by the two component viruses being present singly or in combination. The early literature on mosaic is reviewed in the papers by Stace-Smith dealing with the resolution of the virus complex (*273*, *274*, *275*).

Symptoms, general.—Unfavorable weather conditions, especially late spring frosts, sometimes produce a mottling on the leaves of the fruiting canes that closely resembles mosaic (*22*). However, the leaves of young canes produced in late spring usually appear normal. Powdery mildew will also mimic the symptoms of mosaic in the leaves. However, the powdery white growth of the mildew fungus and the water-soaked areas where it occurs (see fig. 21) can usually be seen when the lower side of the leaf is examined. Leaf symptoms produced by infestation by red spider (mite) or the aphid *Amphorophora rubitoxica* Knowlton (*272*), or those produced by spraying with certain fungicides or through a deficiency of soil boron (*216*), may also resemble raspberry mosaic symptoms.

Symptoms on red raspberries.—A group of diseased plants of a susceptible variety is noticeable because of the short canes, the weak growth, and the sparse yellowish-mottled foliage (fig. 7). Mosaic often causes progressive stunting, the new growth from diseased plants each year being shorter than that of the previous season. Leaves are dwarfed, mottled, and sometimes misshapen. Fruit from a thoroughly infected bush is usually worthless, being dry and seedy, or sometimes crumbly, and lacking flavor (*78*). The character of the foliage on young suckers furnishes the most constant symptom of raspberry mosaic. In late spring, the leaves begin to show large irregular green blisters that arch upward; the tissue around the blisters is yellowish (fig. 7). Leaves produced in very hot weather do not show mosaic pattern or show it very faintly as a flecking of yellow in the normal green blade tissue. In late summer, the leaves near the tip

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FIGURE 7.—Mosaic of red raspberry, showing blisters on a leaf of a new cane late in the spring.

of the sucker show a fine yellowish speckled mottling (fig. 8). Leaves on the fruiting canes are smaller than normal and, on many varieties, show large blisters and yellowish specks.

In many red raspberry varieties the two strains of mosaic virus may occur singly or together and not produce any striking symptoms. The fruit yield and the number and height of canes may be decreased as compared with those of healthy stocks of the same variety (*78*). In some of these cases a faint leaf mottling may be detected in the spring.

Symptoms on black and purple raspberries.—The tips of newly infected black or purple raspberries may bend down, turn black, and

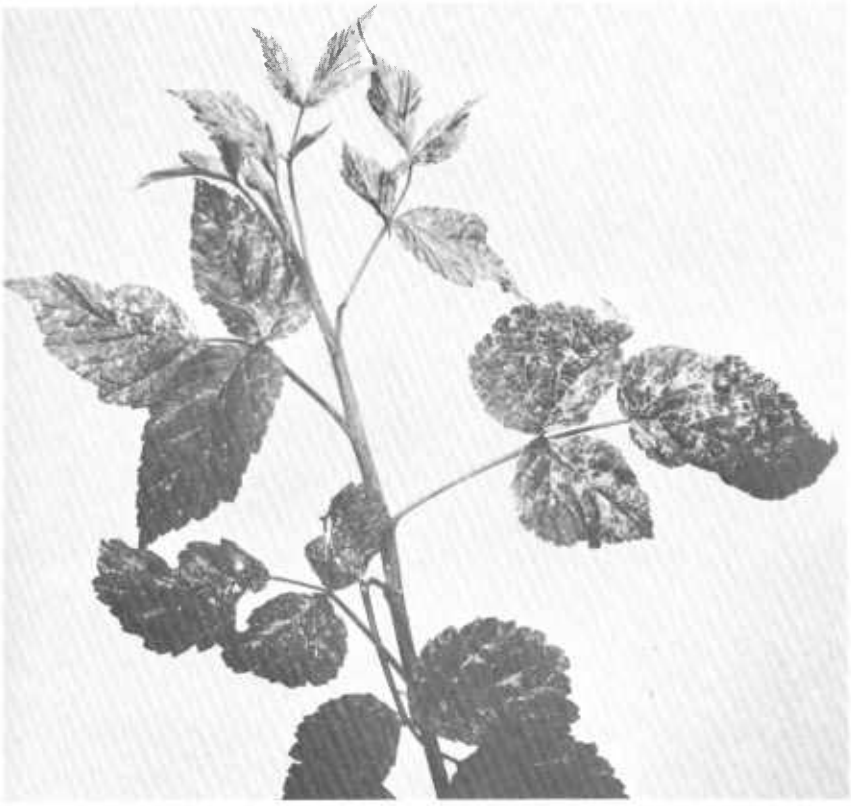

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FIGURE 8.—Mosaic of red raspberry, showing speckled mottling of tip leaves of a new cane late in the summer.

die (fig. 9). Leaves become mottled and develop a pattern of elevated dark-green areas and surrounding light-green depressions. Plants become dwarfed, and shoots tend to be brittle near the tips and to snap off. Foliage produced during hot weather may be almost symptomless, but shoots developing in cool weather will be mottled. The leafing out of mosaic-infected black raspberry plants in the spring occurs a few days after healthy stock has begun to leaf out (*87*).

Black or purple raspberries produced on plants infected with mosaic tend to be dry and seedy. Environmental conditions and some disorders of unknown nature (p. 31) may also cause the fruit to be dry and seedy, but not year after year. (See also the section dealing with mild streak (p. 5), which causes berries to be small and insipid.)

The viruses.—The viruses involved in the raspberry mosaic complex were found by Stace-Smith to belong in two groups (*275*). One group survives prolonged periods in the host at a constant temperature of 37° C. and the other does not (*70*, *276*).

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FIGURE 9.—Tip necrosis of black raspberry caused by a heat-labile component of raspberry mosaic.

The heat-stable group Stace-Smith called rubus yellow-net virus (RYNV) (*276*). Bennett (*22*) described a disease, yellow mosaic, which probably belongs in the rubus yellow-net group. Yellow mosaic produces a yellowish cast on infected leaves of red or black raspberry that persists through hot weather and is not accompanied by blistering or mottling (fig. 10). Stace-Smith and Miss Mellor (*281*) transmitted RYNV to *Fragaria vesca* L. clones, causing necrosis or death. Frazier et al. (*127*) felt that on the basis of the similar symptoms caused in *F. vesca* by RYNV and the necrotic shock virus of strawberry, more work should be done to relate or distinguish between these two viruses.

The heat-labile group, which seems to be composed of numerous strains (*276*), Stace-Smith called black raspberry necrosis virus (BRNV) (*274*). Converse (*78*) later suggested calling this group heat-labile mosaic components (HLMC) (see fig. 9), and such designation is used in this publication.

Raspberry mosaic, defined in the sense of Stace-Smith (*275*) as caused by a virus complex composed of rubus yellow-net virus and heat-labile mosaic components, is found also in Great Britain (*57*), where earlier it was described as veinbanding disease (*51*). As in the United States, the insect vector in Great Britain is *Amphorophora rubi* (Kltb.). In Great Britain there are a number of other viruses transmitted by *A. rubi* (*49*, *52*, *53*, *54*, *60*) that have not yet been

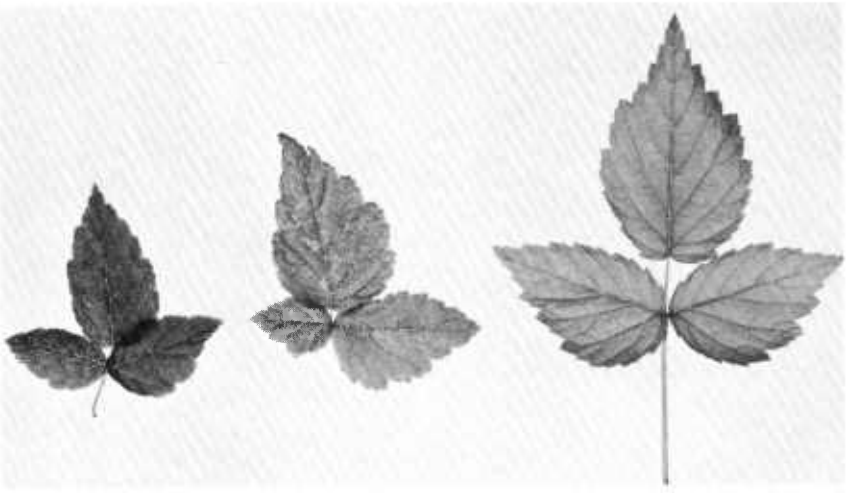

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FIGURE 10.—Rubus yellow-net of black raspberry; on right, healthy leaf.

reported in the United States. (See also p. 18.) The term "raspberry mosaic" has often been used in Great Britain (*69, 148*) to include a number of other aphid-borne viruses besides RYNV and HLMC.

In the raspberry mosaic complex (*sensu* Stace-Smith) strain differences among the virus components probably account for much of the symptom variability expressed in red raspberries (*78*). In relatively tolerant varieties, plants that are entirely symptomless may harbor viruses of the mosaic complex (*274*).

Indicator plants more sensitive to members of the mosaic complex than commercial varieties have been used to detect the viruses, by stem-approach grafting (*49*), leaf-insert grafting (*78*) and aphid transmission (*49, 170, 273*). Black raspberry (*49, 173, 273*) and *Rubus henryi* Hemsl. & Kuntze (*34, 49*), a Chinese species, have been widely used as indicator plants. Of the two, *R. henryi* is the more sensitive under Maryland greenhouse conditions (fig. 11). Studies of the reliability of the leaf-graft method of detecting HLMC showed that three infected leaflets from a source plant left intact in a *R. henryi* test plant for 13 days resulted in infection 95 percent of the time (*79*). *R. albescens* Roxb., *R. saxatilis* L., *R. procerus* P. J. Muell., and certain cultivars of *R. idaeus* L. have also been used as indicators (*49, 273*).

The United States Department of Agriculture has assessed stocks of the major raspberry varieties in the country as to their mosaic-free condition, through leaf grafting on *Rubus henryi* (*80*). Heat-treatment procedures (*34, 70*) were used to purify several of these varieties. The mosaic-free stocks grown in several field locations in the Eastern United States for 1 or 2 years had a final overall level of mosaic infection of 3.3 percent (*80*).

Disease cycle.—The importance of disease-free planting stock in preventing the spread of viruses cannot be overemphasized. Many raspberry varieties (for example, Black Hawk, Chief, Columbian,

Cuthbert, Latham, Newburgh, and Taylor) are very likely to be almost universally infected with viruses of the raspberry mosaic group (*76*, *86*, *274*), and may serve as sources of mosaic for adjacent healthy plants.

The aphids connected with virus movement in *Rubus* have been recently reviewed (*194*). A large raspberry aphid (*Amphorophora rubi* (Kltb.)) is the most common vector of raspberry mosaic (*22*). All stages of the aphid except the egg are viruliferous (*24*). The insects are able to acquire the component viruses of raspberry mosaic after feeding for less than an hour on an infected plant (*273*, *274*). The viruliferous aphids can transmit mosaic to a healthy raspberry plant by feeding on it for just a few minutes (*273*, *274*). The aphids lose their virus charge after feeding on a succession of healthy raspberry plants for 1 to 2 hours (*273*, *274*), or lose it when not feeding after 1 to 4 days depending on the temperature (*276*). This aphid is common on tips of wild red raspberry, many cultivated red raspberries, wineberry (*R. phoenicolasius* Maxim.) (*75*), and some blackberries (*81*). *A. rubi* does not colonize other than *Rubus* plants.

If black and purple raspberries are planted near aphid-infested red raspberry or other *Rubus* hosts that have mosaic, the aphids will colonize the black and purple raspberries and infect them with mosaic. The insects are most common in June and July but are present throughout the growing season (*171*, *334*). Another raspberry aphid (*Macrosiphum* (=*Amphorophora*) *rubicola* (Oestl.)) is of some importance in spreading mosaic in some areas (*24*). The raspberry cane aphid (*Amphorophora sensoriata* Mason) occurs in large colonies on the lower portions of the stems of black and purple raspberry, but it

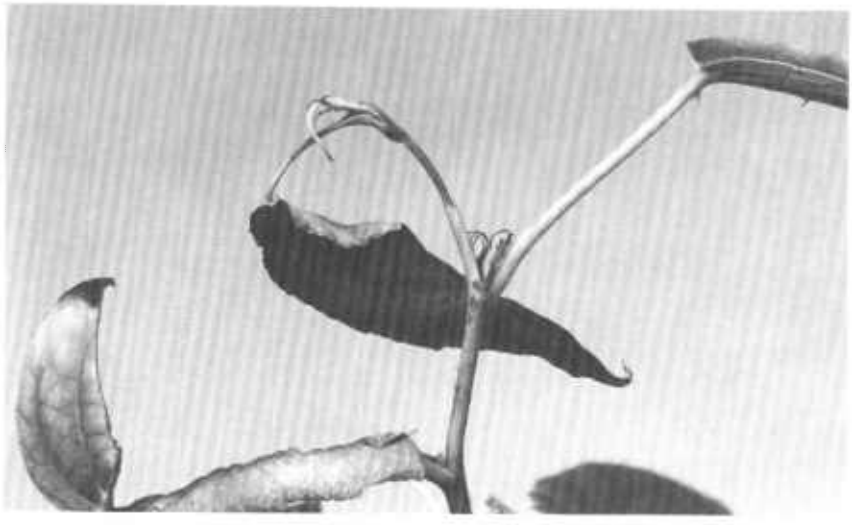

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FIGURE 11.—Necrosis of the shoot tip of *Rubus henryi* caused by one of the heat-labile components of raspberry mosaic introduced into the plant by leaf-grafting. (Note wrapping for leaf graft at lower left.)

does not readily transmit mosaic or other known viruses (*24*, *168*, *194*).

Viruses of the mosaic group as well as other *Rubus* viruses transmitted by *Amphorophora rubi* in Great Britain are reviewed by Cadman (*57*).

Host resistance.—The feeding and colonizing habits of *Amphorophora rubi* on *Rubus* species and on cultivated raspberry varieties have been studied in the United States (*81*, *171*, *260*); in Canada (*96*, *277*); in Sweden (*231*); and in England, where three biological races of the aphid are known to be distinct in varietal preference from any in North America (*42*). The genetics of resistance to *A. rubi* in red raspberry has been studied (*198*, *200*), and the whole subject reviewed (*200*).

Certain red raspberry varieties, as September and Milton, are rather resistant to mosaic infection by aphid transmission, although they will support aphid colonies. Others, as Canby and Indian Summer, may be generally free from mosaic because the large red raspberry aphid does not usually live on them (*81*). The British red raspberry variety Lloyd George, on which North American *Amphorophora rubi* do not colonize (*81*, *96*, *171*, *277*), has been used successfully in transmitting resistance to colonization to other varieties in several but not in all cases (*81*, *96*, *277*). Several other sources of genetic resistance to colonization by *A. rubi* in North America have also been noted (*81*, *96*, *171*, *260*, *277*). The red raspberry variety Ranere (St. Regis) is unusual in that it harbors the component viruses of raspberry mosaic but *A. rubi* is unable to transmit them to other suscepts (*277*).

Most purple and black raspberries are susceptible to mosaic and to aphid vector feeding, but usually differ in their ability to tolerate mosaic. New Logan, Bristol, and Black Hawk black raspberries appear to tolerate the components of mosaic better than the Cumberland black variety. Heritable differences in raspberry innate resistance to mosaic have been demonstrated (*187*). Blackberries are sometimes infected with raspberry mosaic, usually in symptomless condition.

Control.—Use of mosaic-free stock is necessary to establish a vigorous raspberry planting. Mosaic-free stocks have, in some cases, outyielded mosaic-infected stock severalfold (*78*). Mosaic-free stocks of many desirable raspberry varieties exist (*80*), but some important varieties are not at present known to occur free from mosaic. Mosaic-infected varieties may be grown with profit, but it is desirable to keep them isolated from mosaic-free stock. As a general rule, it is well not to plant red raspberries near black raspberries, unless both are carefully sprayed for aphid control, even though both are mosaic free. This is because black raspberries are more susceptible to mosaic, and red raspberries are prone to harbor a large number of vector aphids. Every effort should be made to obtain stock certified virus free, as well as free from systemic bacterial or fungal diseases.

Roguing a raspberry planting is essential to its continued health. Roguing for mosaic is best accomplished when symptoms are most clearly seen—in late spring during cool, cloudy weather and in the fall. Infected plants should be destroyed in such a way that the

virus-carrying aphids present are not scattered throughout the field. Scorching infected plants in place with a blowtorch or a weed-control flamethrower (*266*) or applying a good aphicide before roguing is recommended.

Removal or marking those black raspberry plants that are later than the rest to leaf out in the spring is advisable, as they are likely to have raspberry mosaic (*87*).

Roguing in fields where mosaic is present in more than 5 percent of the plants is probably uneconomical because of the high likelihood of the existence of symptomless as well as symptom-producing viruses in the stock. Such fields are generally maintained only as long as they produce enough fruit to be profitable, before being plowed out.

Elimination of wild *Rubus*, particularly raspberries and wineberries, around commercial plantings will reduce the source of mosaic and aphid vectors coming into the planting. Aphids are present throughout the growing season, but are most numerous in early summer (*171*, *334*). The use of insecticides effective against aphids, such as malathion and parathion, will greatly reduce the number of aphids (*78*). The standard precautions, which appear on the manufacturer's label, as to the timing of applications and the care and handling of the dangerous chemicals should be closely followed. Complete eradication of aphids is difficult, however, for winged aphids from a distance arrive in sprayed fields and may infect a few plants before being killed by the aphicide. Thus, even in fields sprayed for aphids, continual roguing must be practiced to remove new mosaic infections brought in by the aphids.

Ultimately, because of the buildup of latent viruses, the plantings will need to be plowed under and replanted with mosaic-free stock more recently propagated from indexed sources.

One of the most promising control methods of raspberry mosaic is through plant breeding. Several red raspberry varieties that are now grown commercially in the United States are either resistant to *Amphorophora rubi*, the major vector of the mosaic viruses, or possess innate resistance to the viruses themselves. Resistance to mosaic should be one of the major objectives of any raspberry breeding program in the United States.

Other Raspberry Virus Diseases

Several raspberry virus diseases that have not yet been noted in the United States are reported from Canada and Great Britain. Vein chlorosis virus occurs in red raspberry; it is transmitted by *Aphis idaei* V.d.G. but not *Amphorophora rubi* in Great Britain (*50*) and British Columbia (*278*). In Britain, three strains of the raspberry vein chlorosis virus are recognized (*50*). In British Columbia, the virus is heat stable at 37° C. in the host plant and can occur in the host in the presence of HLMC (=BRNV) (*278*).

Several other virus diseases of raspberry in Great Britain that have *Amphorophora rubi* as a known or a probable vector have been studied by Cadman (*49*, *52*, *53*) and reviewed by him in 1961 (*57*). Among these virus diseases veinbanding appears to fit into the group of viruses belonging to the raspberry mosaic complex in North America

(*57*). The relationships between the British virus diseases raspberry leaf mottle (*49*), raspberry leaf spot (*53*), yellows (*52*), bushy dwarf disease (*57*), and curly dwarf disease (*57*) have not been worked out with the North American *Rubus* viruses. The viruses causing raspberry leaf mottle and raspberry leaf spot are heat labile while the viruses causing yellows and veinbanding (probably RYNV) will endure 40° C. for 60 days in the host plant (*70*). Heat-labile viruses of the group transmitted by *A. rubi* and other viruses of the group listed above have been mechanically transmitted to *Chenopodium* spp., where they caused local lesions and systemic symptoms (*57*, *58*).

The presence of cucumber mosaic virus in raspberry has been suggested in Pennsylvania (*359*) and demonstrated in Britain (*149*). This seems to be rare.

Three soil-borne viruses (table 1, after Cadman (*57*)) are known on raspberry in Britain, and the considerable literature in this field

TABLE 1.—*Some properties of nematode-borne viruses that infect raspberry in Great Britain*

[After Cadman (*57*)]

Disease	Virus	Size of isometric virus particles	Thermal inactivation point	Nematode vector	Reference
		mμ	° *C.*		
Raspberry yellow dwarf.	Arabis mosaic.	29–30	58–61	*Xiphinema diversicaudatum* Micol.	*150*, *154*, *155*.
Scottish raspberry leaf curl.	Raspberry ringspot.	29–30	66–70	*Longidorus elongatus* (de Man).	*151*, *155*, *297*.
Unnamed----	Tomato black ring, beet ring-spot strain.	29–30	58–62	*Longidorus elongatus* (de Man).	*152*, *153*, *155*, *268*.

has been reviewed by Cadman (*57*). They are transmitted by various ectoparasitic nematodes (table 1) and through the seed and pollen. The wide host ranges include weeds (*207*). Marked differences exist in varietal susceptibility to these viruses (*57*). In the United States soil-borne viruses have been recognized in cultivated raspberry and blackberry in California (*4*, *193*). (See p. 60.) Stace-Smith has described a ringspot virus on red raspberry in British Columbia (*280*) that fits in this group (fig. 12). This virus is serologically related to tomato ringspot virus (*217*).[5] Raspberry ringspot is definitely nematode transmitted [5] and has been transmitted experimentally to straw-

[5] Stace-Smith, R. Personal communication, 1964.

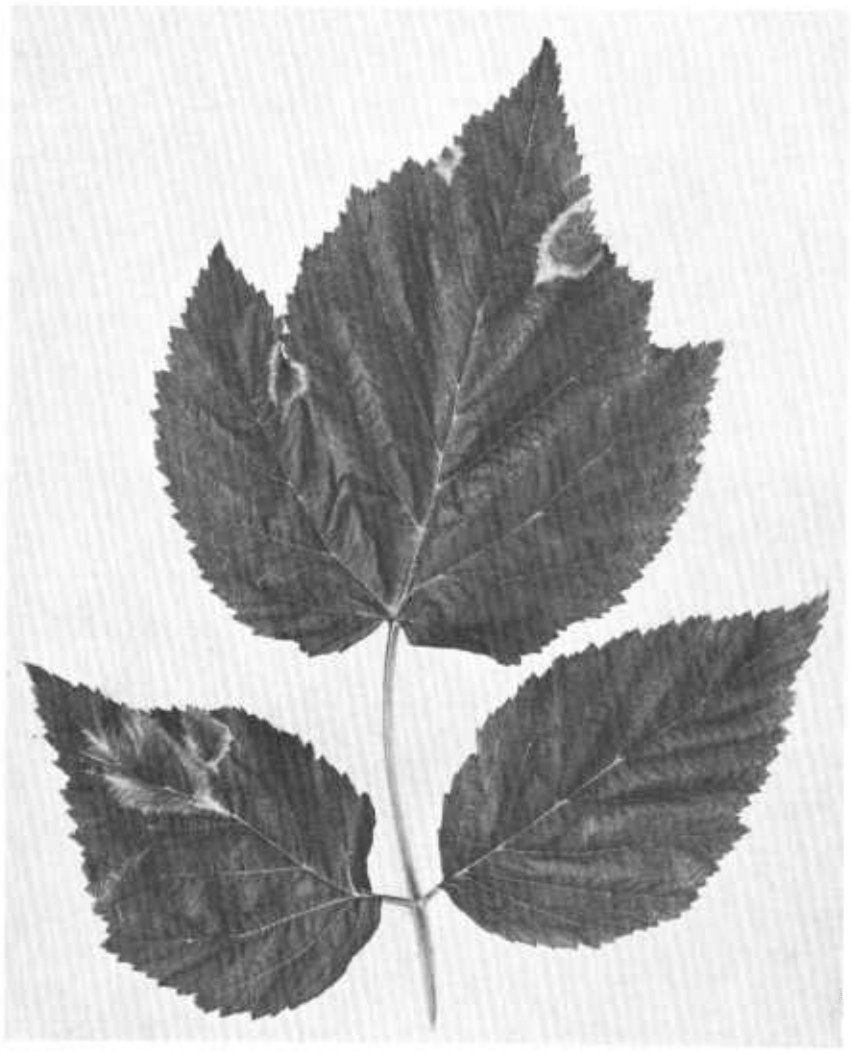

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FIGURE 12.—Raspberry ringspot of red raspberry. (After Stace-Smith, 1962.)

berry (*217*, *316*), where it is seed transmitted (*217*). A similar, perhaps identical, disease for which the mode of spread is unknown occurs in Oregon and Washington (*313*, *316*). Yellow blotch-curl, described in Ontario by Chamberlain (*67*), may belong in this group of viruses, as well as raspberry decline in Oregon (*356*).

Raspberry decline, a viruslike graft-transmissible disorder in Oregon, characterized by recurving of leaves, crumbliness of berries, and a progressive weakening of the tops and roots of infected plants, was considered by Zeller and Braun (*356*) to be soil related.

Rubus stunt occurs in Britain and in Europe (*123*, *243*) on raspberry and blackberry and is transmitted by the leafhopper *Macropsis fuscula* Zett. (*123*, *124*), which has been recently reported in British Columbia (*18*). Rubus stunt is not known in the United States. The virus is heat labile in *Rubus* (*301*). (See also p. 65.)

Experimental infection of raspberry by tobacco mosaic virus was reported (*115*), but this early work needs confirmation. Tobacco necrosis virus and occasionally tobacco rattle virus occur in *Rubus* roots (*57*).

Fungus Diseases

Anthracnose [6]

Anthracnose is one of the most destructive fungus diseases wherever black and purple raspberries are grown. Over the United States the average annual losses from anthracnose are considerable. On the other hand, although the fungus is common on red raspberry, it is not usually a serious disease of this bramble. Little correlation between red raspberry yield and anthracnose incidence was found in Nova Scotia (*2*).

Key historical papers on etiology, symptomatology, pathological histology, and control of anthracnose on raspberries are by Burkholder (*45*) and Jones (*189*).

Symptoms.—The most striking symptoms of anthracnose of black and purple raspberries are on the canes, the older ones of which are frequently found spotted with light-grayish areas that are 3 millimeters or more in diameter. Unsprayed Cumberland black raspberries averaged 173 lesions per cane in a Michigan test (*135*). On young shoots the disease first appears as very small, slightly sunken, circular purple spots. As the disease progresses, the spots enlarge and become pale buff or ash gray at the center, the margins somewhat raised and purple in color (fig. 13). The older lesions extend into the woody portions of the canes. As the infected black or purple raspberry canes dry out, they may crack up and down, for an inch or two. In some cases the old canes present a rough appearance, sometimes warted or knotted, because of the swelling of tissue beneath the bark in the regions affected by the fungus. Canes with many cankers may be partly girdled, and the fruit produced on such canes the following season may fail to develop to normal size, often shriveling and drying up, especially in seasons of drought. Such canes are prone to winter injury. A late infection may occur on the lateral branches, stunting them and preventing proper growth and bud formation for the next season.

On red raspberries the cane symptoms include grayish lesions, which are usually very small; they are smaller and much less abundant than on black or purple raspberries. The lesions may be either sunken or raised. The most common manifestation of the disease is that of extensive grayish superficial growth on current-season canes. This is most conspicuous on the side exposed to the sun. The gray appearance

[6] Caused by *Elsinoë veneta* (Burkh.) Jenkins; imperfect stage is *Sphaceloma necator* (Ell. & Ev.) Jenkins & Shear.

BN–26735

FIGURE 13.—Anthracnose of raspberry canes: *A*, Older cane of black raspberry; *B*, young purple-raspberry cane.

is due to the uniting of many small surface colonies of the fungus that develop on the current-season canes during late summer or early fall. Colonies of reddish pimplelike acervuli arranged in irregular concentric rings are often formed on the gray bark (fig. 14). This graying of infected red raspberry stems is commonly referred to as "gray bark" and is probably of more importance as a source of inoculum than as a cause of direct damage to the host.

Anthracnose fungus sometimes attacks the leaves of red, black, and purple raspberries but rarely defoliates the plants. The irregular spots, at first very small, may later become 1.5 millimeters in diameter. They have a light-gray center and a purple margin. The diseased tissue frequently drops out, so that shotholing results. The petioles are also subject to attack. Spots on the leaves caused by an

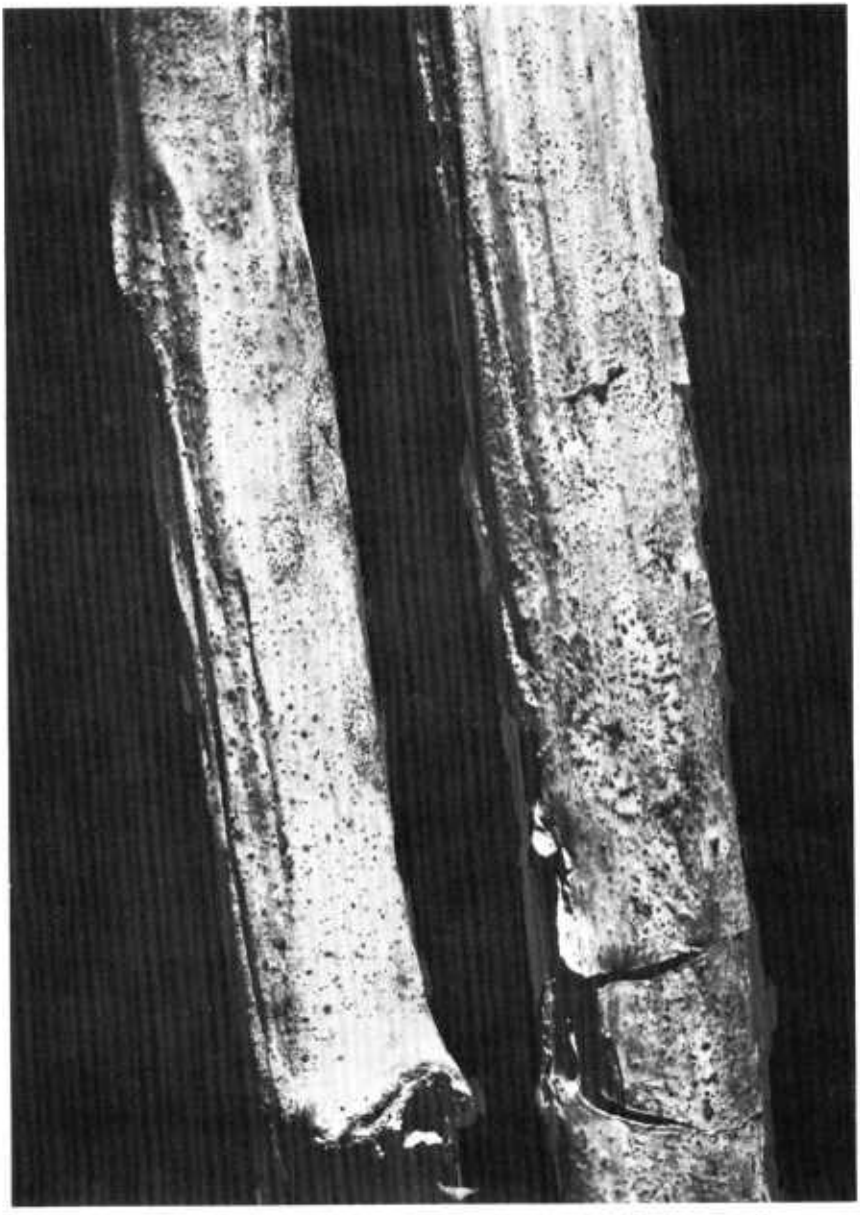

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FIGURE 14.—Anthracnose of Latham red raspberry canes showing the gray-bark stage.

entirely different fungus (*Sphaerulina rubi* Demaree & M. S. Wilcox) are often mistaken for anthracnose but can be recognized by their

distinct white centers and more definite margins (see p. 35 and fig. 19, *A*). The control of these two kinds of leaf spot is much the same, however.

When the branches bearing the berry clusters are badly infected, especially in black and purple raspberries, the fruit fails to mature properly, particularly in dry weather, ripening without pulp and becoming dry and hard. The fungus may also attack the peduncles and the fruit pedicels girdling them. Yield is most seriously affected by this type of infection (*131*). In rainy weather drupelets of the berry may become infected, causing them to become rusty brown and scabby.

Causal organism.—*Elsinoë veneta* ascocarps (*184*) are produced on stromatic cushions about 75μ in diameter on overwintering cane lesions. Several isolated asci are formed in the ascocarp and are globose, thick-walled, 24μ to 30μ. Ascospores are hyaline, 4-celled, ovate, slightly curved, 18μ to 21μ x 6.5μ to 8μ, borne eight per ascus in rows. Ascospores are forcibly discharged from asci when the ascocarp weathers away. Germinated ascospores form secondary spores by budding that are identical with the conidia.

The acervuli of the *Sphaceloma necator* stage are formed on subcuticular fungal pseudoparenchyma. Short, unbranched conidiophores arising from the acervuli bear the hyaline, oblong, 2-guttulate, nonseptate conidia, 5μ to 7μ x 2.5μ to 3μ, which are held together in a mucilaginous substance (*45*).

Races of the fungus are not recognized.

Disease cycle.—The anthracnose fungus does not become systemic in infected plants. Individual lesions spread out from their original points of infection only slightly. Succulent, rapidly growing parts are susceptible, while older, hardened parts are only superficially infected (*45*).

About a week after inoculation with conidia, small purple spots begin to appear on new leaves. Upon the death of the cells at the center of a spot, the part turns ash gray, and the pimplelike acervuli are formed. The fungus attacks the young growing ends of canes and branches and invades the living tissue of the bark, causing the death of cambium and phloem cells. The conidia produced on acervuli are dispersed by drops of dew or rain. During the growing season the fungus is widely spread throughout a plant and to new plants by means of conidia. Moisture favors the development of infection (*33*).

The fungus lives over winter in the canes and gradually matures not only a new crop of conidia in the spring but also may produce the overwintering ascigerous stage, which is comparatively rare, however, in the United States (*46*). Conidia and ascospores production in the spring coincides with the leafing out of brambles (*45*). Ascospores are found in the air only when the relative humidity is high for long periods (*175*). The factors determining formation, longevity, and release of conidia and ascospores and their successful invasion of host tissue are not well understood. Survival of the fungus in *Rubus* debris requires more investigation.

Host resistance.—The anthracnose fungus occurs only on wild and cultivated *Rubus* species. A number of wild species are resistant.

Most varieties of black and purple raspberries, however, are very susceptible. Black Hawk and Dundee are more resistant than other black raspberry varieties. Anthracnose resistance in black raspberries probably involves more than one gene (*2*, *72*). Sodus and Marion purple raspberries are more resistant than other purples. Although the red variety Latham is susceptible to anthracnose, red raspberries generally are not as severely damaged as are black raspberries. Resistance to anthracnose in red raspberry is somewhat related to lack of spines and pigment in the canes, and presence of wax on the canes. However, anthracnose resistance seems to be more related to physiological than to morphological differences (*186*). The nature of this physiological resistance is not known.

Control.—A planting site should be chosen that has good air drainage. A new planting set out with disease-free nursery stock is likely to remain relatively free from anthracnose for several years (*37*).

The mycelium of the anthracnose fungus can live over winter in the old cane parts (*45*). The stubs of old canes (commonly called handles), usually left attached to black raspberry tip plants, may become sources of new infection. For this reason the part of old canes remaining above ground after planting should be removed and burned. Tip plants obtained from the grower's own field should be planted while dormant, before new growth becomes infected from nearby infected canes.

All fruiting canes should be cut out and burned after harvest. New canes that appear to be badly infected at this time should also be destroyed. Healthy canes should be thinned out sufficiently to allow good air drainage.

Thorough cultivation should be given the crop, especially from budbreak until July, to insure that the rows are kept free from weeds and rubbish, which tend to keep the canes from drying out, may provide inoculum, and may interfere with good spray coverage. Because anthracnose conidia may be carried from wild brambles to field plantings by insects or by driving rain, wild brambles in nearby fence rows and hedges should be eliminated so far as practicable.

Anthracnose is not hard to control if adequate cultural practices and spray schedules are used *before* the disease becomes well established. Spray applications protect only the uninfected parts of the plant.

Two or three applications of spray properly timed will generally satisfactorily control cane and fruit infection by the anthracnose fungus (*135*). The first application of fungicide must be made during early spring when the leafbuds are beginning to open and new leaves are exposed one-half to three-fourths of an inch. Spores are being liberated from the old infected canes about this time and will be killed by the spray material. Lime-sulfur has been the preferred material, but sodium dinitro-o-cresylate (DNOC) has also been widely used for this first application. All sides of the canes should be thoroughly wetted with the spray mixture. Timing is critical, as the anthracnose fungus is resistant to the fungicide in fully dormant lesions, and spraying unfolded leaves may cause injury. One properly timed lime-sulfur spray reduced the incidence of anthracnose by 68 percent in a Michigan test (*135*).

A suitable period for a second application is about the time the first flowerbuds are visible and new canes are about 6 inches high. Where anthracnose is severe, a third spray applied after petal fall when new canes are 12 to 15 inches high is advisable. Captan or ferbam has been used for the second and third spray applications. Addition of a wetting agent to the spray mixture is suggested by many workers. Where possible, these sprays should be applied *before* a predicted rain.

Cane Blight [7]

The fungus causing cane blight is able to infect the canes only through wounds, especially pruning wounds. Cane blight is found in all raspberry growing regions. The disease does not usually cause as severe damage as anthracnose or spur blight; with these it is often associated and confused. Because the fruiting bodies of the cane blight fungus develop only on dead portions of living canes, the fungus is often blamed for winter injury and other environmental damage. The first comprehensive study of the fungus was by Stewart and Eustace in 1902 (*285*).

Symptoms.—On new canes lesions develop at wounds. A brown to black infected area develops that may extend down one side, or ring the cane for several inches below a wound. Fruiting canes infected the previous season have light-colored fissured bark covered with the small black fruiting bodies of the fungus (fig. 15). The infected area often extends over several nodes. Spur blight lesions, in contrast, are usually limited to separate nodes (see p. 42 and fig. 23). The wood of canes infected with cane blight is brown and breaks easily. Lateral shoots on infected canes grow poorly and often wilt and die in warm weather. This collapse of fruiting canes is the first serious effect of the disease usually noticed by growers. Numerous small black pycnidia of the cane blight fungus are visible on infected new canes in the summer. The numerous spores produced by these pycnidia exude over the bark and give it a characteristic gray smoky appearance (*5*).

Causal organism.—Perithecia of *Melanomma coniothyrium* (*166*) form on dead bark of *Rubus* fruiting canes and in many other hosts in the fall. Perithecia are 250μ to 300μ, black, subglobose to a little flattened with minutely necked ostioles, and are formed subepidermally. The clypeus of the perithecium is well developed with a clear ostiole in the center. Perithecial walls are of the same thickness throughout. Asci are cylindrical, stipitate, 8-spored, 70μ x 6μ. Ascospores are subbiseriate in the ascus, 4-celled, obscurely guttulate, constricted at the septa, fusiform, brown, 12μ to 15μ x 3.5μ to 4μ (*166; 255, vol. 2, pp. 29–30*). Pycnidia are superficial, dark, 180μ to 200μ. Conidia are olivaceous, globose to elliptical, 2.4μ to 5.0μ x 2.0μ to 3.5μ (*255, vol. 3, pp. 306, 486*). Conidia are exuded through ostioles in gelatinous masses. The cane blight fungus is widely known as *Leptosphaeria coniothyrium*. The transfer to the genus *Melanomma* was made by Holm in 1957 (*166*).

[7] Caused by *Melanomma coniothyrium* (Fckl.) L. Holm (=*Leptosphaeria coniothyrium* (Fckl.) Sacc.); imperfect stage is *Coniothyrium fuckelii* Sacc.

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FIGURE 15.—Raspberry cane blight infection on a fruiting cane. Note discolored area and fruiting bodies. (After Fulton, 1960.)

Disease cycle.—Pycnidia on *Rubus* cane debris continue to release conidia for at least 4 years (*132*). These conidia probably constitute the primary spring inoculum, although the ascigerous stage is also functional. The organism is predominantly a wound parasite on canes, although Koch (*201*) presented good experimental evidence for the infection of healthy leaflets.

The following are important infection courts for the cane blight fungus on *Rubus* in the United States: Insect wounds, such as those

made by the snowy tree cricket, *Oecanthus niveus* (DeGeer), mechanical abrasion of canes caused by their rubbing together in the wind, and the cuts made when canes are headed back (*25*). In Europe the cane blight fungus readily invades the feeding wounds made by the raspberry cane midge (*Thomasiniana theobaldi* Barnes) (*238*), an insect not occurring in the United States.[8]

Conidia or ascospores can quickly germinate and establish infections in cane wounds at any time during the growing season under moist conditions. Warm rainy weather in midsummer followed the next year by a wet spring and a dry fruit-ripening period lead to the greatest crop losses from cane blight in black raspberry, because of the collapse of fruiting canes infected the previous year (*25*). The fungus continues to invade and sporulate on the young infected canes, more rapidly in already weakened than in vigorous canes. The mycelium overwinters in cane lesions and produces perithecia and additional pycnidia the following spring. Apparently the fungus does not invade uninfected dead, overwintered canes as a saprophyte, although the fungus that has infected the living cane sporulates in the area of infection when the cane has died. New infections occur only on wounded first-year canes during the growing season. Saprophytism of *Melanomma coniothyrium* has not been well studied, however. *Melanomma coniothyrium* is the principal secondary invading organism in the cane lesions caused by *Phragmidium rubi-idaei* (DC.) Karst. in the Pacific Northwest (*349*, *351*). In Great Britain but not in the United States another weak pathogen, *Cryptosporium minimum* Laub., causes a minor disease known as black blotch on mechanically damaged bramble canes (*147*).

Host resistance.—All commonly grown varieties of raspberry are susceptible to cane blight damage. Wild red raspberry is susceptible (*5*). The fungus also causes diseases of rose, apple, pear, strawberry, and other plants, but more exact taxonomic and host-range work is needed here (*166*).

Control.—Care in pruning is the best control for cane blight. Experimentally cane blight was reduced 94 percent in Michigan by immediately treating pruned raspberry canes with a wound dressing (*132*). Plants should be pruned at least 3 days before an anticipated rain so that pruning wounds will callus before infection can develop (*134*). Old infected canes and infected tips serve as sources of spores of the cane blight fungus for several years (*132*) and should be routinely cut out and hauled away or burned. Pruning and cultivation systems that allow good air drainage among the plants reduce the amount of infection. Spraying with fungicides has not given satisfactory control of cane blight.

Fruit Rots

Raspberry fruits are among the most perishable of the fruit crops. Warm wet weather at harvest favors the development of fruit rots (*226*). Many different fungi cause raspberry fruit rots. The principal fruit rots are Alternaria rot, blue mold rot, Cladosporium rot, gray

[8] Foote, R. H. Personal communication, 1961.

mold, and Rhizopus rot (*156*, *336*, *344*). All are widespread and develop fastest on overripe and bruised berries. Gray mold is the most common fruit rot in the field and Cladosporium and Rhizopus rots the most common in storage (*15*, *336*). Other fungus diseases and disorders occur on fruits and warrant some comment. General preventive measures applying to all five of the major fruit rots will be considered. U.S. Department of Agriculture Handbook No. 189 deals with fruit rots of brambles and other small fruit crops and grapes (*156*).

Alternaria rot.[9]—Alternaria rot occurs in mature raspberry fruits, usually after harvest (*15*, *344*). The infected fruits are covered by a dark-gray mycelium on which chains of dark, muriform conidia are borne. *Alternaria humicola* Oud. was one of two molds most commonly found on black raspberries harvested in Michigan (*20*, *119*).

Blue mold rot.[10]—Blue mold rot develops on berries in storage. It becomes established first on bruised berries and spreads rather fast (*15*, *156*, *336*). The fungus growing over infected fruit is at first white, then it turns blue green.

Cladosporium rot.[11]—Cladosporium rot is common on harvested raspberry fruit. It produces an olive-green mold growth on stored raspberries, especially on the inside or cup of the fruit, but causes little tissue breakdown (fig. 16, *D*). Infections begin on overripe or bruised berries stored 5 to 7 days at about 40° F. (*156*). In Michigan *Cladosporium epiphyllum* Pers. and *Cladosporium* spp. were commonly found on black raspberries (*20*, *119*). Cappellini, Stretch, and Walton (*65*) found *Cladosporium* spp. to be among the predominant fungi on harvested fruit of Latham red raspberry in New Jersey.

Gray mold.[12]—*Botrytis cinerea*, causing what is known either as gray mold fruit rot or Botrytis fruit rot, also causing a cane canker, is a facultative parasite that attacks raspberry, grapes, and other small fruits and many other crops. Because of its importance wherever raspberries are grown, the fungus will be treated in more detail than the other fruit rot pathogens. The fungus probably develops in dead leaves and plant debris wherever brambles are grown. It commonly causes a watery rot of red raspberry fruits both in storage and in the field, especially in cool, damp weather.

Symptoms.—Infected fruits develop a watery soft rot; in the field or in storage they are overgrown at the same time by a dark-gray, dusty mass of hyphae and conidia (fig. 16, *A*). Cane cankers develop in Scotland and Canada and to some extent in cool, humid parts of the United States. Infected canes exhibit a distinctive "watermark" symptom in the spring (*68*, *165*, *179*).

Causal organism.—Dark branched conidiophores develop over diseased tissue, producing single-celled, ovate, hyaline conidia, 8μ to 11μ x 11μ to 15μ. Small black sclerotia (up to 2 mm. long on canes, formed subepidermally) develop on dead or infected plant parts. These sclerotia produce conidia in the spring. *Botrytis cinerea* is the im-

[9] Caused by *Alternaria* spp.
[10] Caused by *Penicillium* spp.
[11] Caused by *Cladosporium* spp.
[12] Caused by *Botrytis cinerea* Pers. ex Fr.

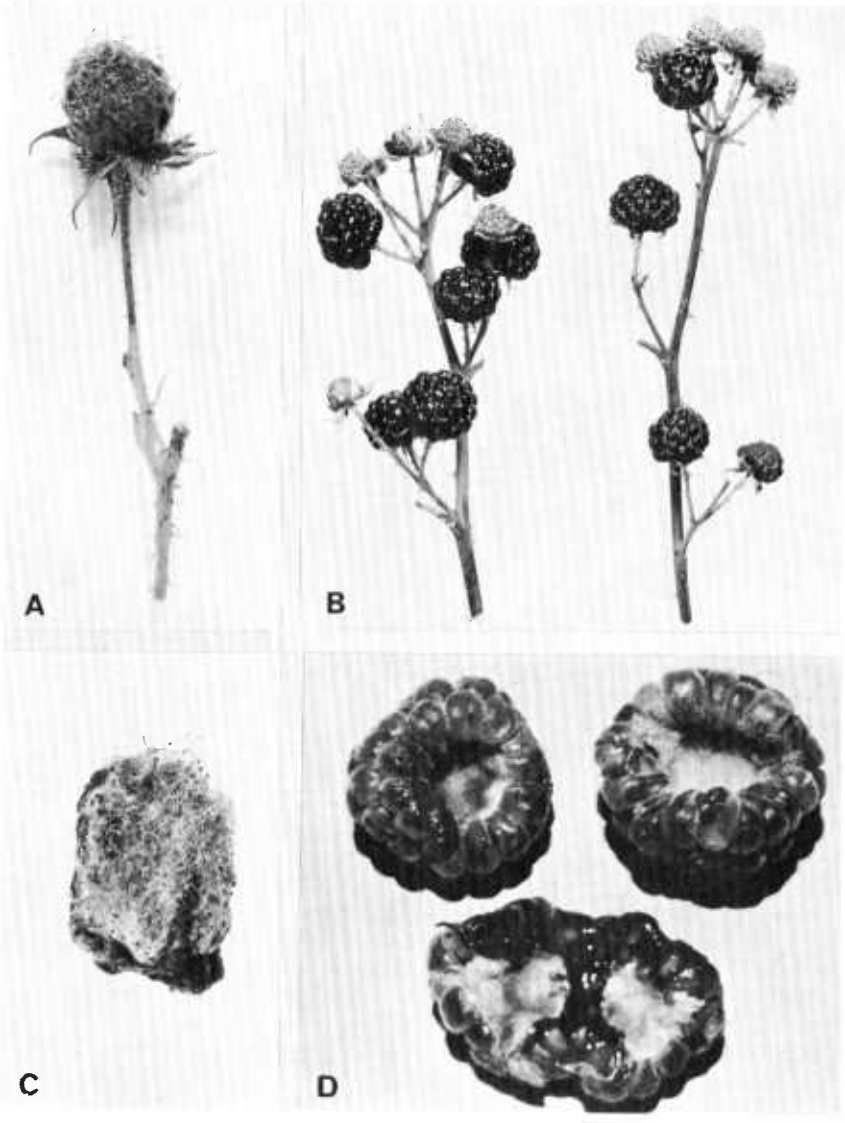

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FIGURE 16.—Raspberry fruit rots: *A*, Gray mold; *B*, Brown berry disorder; *C*, Rhizopus rot; *D*, Cladosporium rot. (Part *D*, courtesy of New Jersey Agricultural Experiment Station.)

perfect stage of several *Botryotinia* (*Sclerotinia*) species; the perfect stage, however, seems to play little part in the life cycle of the fungus on *Rubus* (*142*). Isolates of the fungus from unrelated hosts have broad pathogenicity (*142*, *179*).

Disease cycle.—Conidia formed from overwintering sclerotia, or from overwintering mycelium in cane cankers, are borne by wind or

splashing rain (*177*, *180*) to the suitable substrates, which are often dead or dying plant parts. In Scotland the fungus frequently becomes established in the organs of the very young opening flower. While the flower is young, only a limited amount of mycelium develops, but these quiescent infections become active as the berries ripen. Immature flowers damaged by frost are more heavily infected than uninjured flowers (*179*). Whether quiescent infections commonly develop in the United States is not known. Infected fruit left unpicked and the exposed receptacles, which quickly become infected, that remain on pedicels after sound fruit is picked are major sources of inoculum (*179*).

Host resistance.—Black raspberries are not commonly infected with gray mold fungus (*20*). There are no important sources of varietal resistance to this fruit rot among red raspberries, only different shades of susceptibility. Red raspberry seedlings with hairy, spine-free, nonpigmented canes with moderately dense wax were most resistant to *Botrytis cinerea* in Scotland (*186*). Varieties whose fruit is not bunched together and varieties with quick-drying receptacles should tend somewhat to escape gray mold fruit rot (*179*).

Control.—Sprays of captan for Botrytis fruit rot control on red raspberry after the blossoms are open are successful and are used in New Jersey (*290*); captan and ziram dusts are used at this time in Washington (*333*). Fungicides have been of doubtful value in Scotland, except that very early captan sprays applied as blossoms are just opening have seemed promising (*178*). Frequent, thorough picking is very important in control (*178*, *335*).

Rhizopus rot.[13]—Rhizopus rot is a very common and serious watery soft rot of harvested raspberries that occurs in storage (*15*, *336*, *344*). Bruised and overripe berries are very susceptible to invasion by the *Rhizopus* organism, which produces a dense, coarse, grayish mold over the berries that is soon covered with small black sporangia (fig. 16, *C*). Sound berries in warm storage are quickly attacked by *Rhizopus* from nearby infected berries (*156*).

Other fruit rots.—Anthracnose occurs on raspberry fruit, and occasionally late leaf rust, powdery mildew, the stamen blight fungus, and yellow rust infect them. The symptoms and controls for these diseases are described in separate sections (pp. 21, 32, 39, 81, 52). *Aspergillus* spp. are sometimes involved in storage rots (*15*, *119*, *336*). Also, frequently isolated from surface-sterilized black raspberry druplets that appeared to be sound were *Fusarium*, *Monilia*, *Mucor*, *Oospora*, *Pullularia*, and *Trichoderma* (*119*). Brown berry of black raspberry is a condition of unknown etiology. The berries turn brown and remain hard when they are about half mature (fig. 16, *B*). The disorder is sporadic in the field and is seasonal. No control is known (*99*).

General preventive measures.—The importance of picking only sound, firm fruit to avoid development of fruit rots in harvested berries cannot be stressed too much. Raspberries with a high fruit-rot count are downgraded in price and may even be rejected by processors, who

[13] Caused by *Rhizopus stolonifer* (Ehr. ex Fr.) Lind.

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must maintain a mold-count tolerance on their products (*119*, *120*). The standardized Howard mold count method is used (*10*). Apparently sound fruit picked in hot humid weather may have a high mold count just as it comes from the field (*120*, *226*). The overripe and bruised berries in the pack are more likely to become rotted first, paving the way to the spoiling of sound fruit. Care in picking and handling to avoid bruising is therefore very important (*120*). It is also important to avoid delay in processing and marketing.

The fruit rots develop slower at low temperatures (*283*). Picking is therefore best done early in the morning when the berries are coolest (*335*). They are best stored under refrigeration (32° to 40° F.), or if refrigeration is not available, in the shade with good aeration before quick marketing (*335*). Good air movement around the berries is very important in facilitating heat exchange and in dissipating the heat developed by the normal respiration of the fruit (*270*). Fungicide sprays during the preharvest and harvest periods aid in reducing fruit rots (*21*, *145*, *178*, *266*, *290*) but are no substitute for frequent, thorough pickings and careful handling. Captan as such an aid can be used up to and including the day the fruit is harvested.

Used berry boxes and carriers are often covered with debris and mold spores. Because their repeated use favors rot spread, methods for their decontamination in the field have been studied (*120*). Sodium ortho-phenylphenate was very effective in Washington (*41*) but is not cleared for commercial use on picking boxes. Copper 8-quinolinate has also performed well (*1*, *176*), and 25-percent solution is cleared for use on fruit picking boxes by the U.S. Department of Agriculture (*304*, *p. 154*).

Chemical controls of fruit rots after harvest have been investigated but are not generally used commercially. The sodium salt of dehydroacetic acid gave excellent control of Alternaria, Botrytis, and Rhizopus fruit rots of raspberry in Michigan tests (*344*) and of Alternaria, Aspergillus, Penicillium, and Rhizopus rots of blackberry in Oklahoma (*15*). This substance is not presently cleared for commercial use on raspberries. Effective control of Botrytis, Cladosporium, and Alternaria rots of Latham red raspberries by brief fumigation with sulfur dioxide gas was achieved without injuring the fruit or affecting its flavor (*65*). Nitrogen trichloride gas gave excellent control of Botrytis and Rhizopus rots of raspberries and blackberries in an Oregon test (*312*).

Storage of raspberries in an atmosphere of 30 percent carbon dioxide at 55° to 60° F. and a relative humidity of 80 to 90 percent reduced the amount of decay by one-half as compared with storage under atmospheric carbon dioxide levels (*270*, *336*).

Late Leaf Rust [14]

Late leaf rust (also called autumn rust, late raspberry rust, and late yellow rust) is a relatively minor leaf-spotting disease attacking only cultivated red and purple raspberries, and some wild red raspberries. Late leaf rust occurs mainly in the northeastern quarter of the

[14] Caused by *Pucciniastrum americanum* (Farl.) Arth.

United States in July and August. The life cycle of the rust has been studied by Darker (*89*) and Dodge (*107*).

Symptoms and disease cycle.—Late leaf rust fungus of red and purple raspberry is not systemic, as is the orange rust fungus of black raspberry (p. 36). On mature leaves late leaf rust fungus causes many small spots to develop; these spots turn yellow, then brown, before the leaves die in the fall. Small pustules filled with powdery yellow spores (not waxy like orange rust spores) are formed on the undersides of infected leaves (fig. 17). Badly infected leaves drop prematurely; and the canes of susceptible varieties, as Latham, may be bare by September. Such plants are prone to winter injury. Petioles, canes, and even fruits (fig. 18) are sometimes attacked.

Causal organism.—Uredia are hypophyllous and numerous over large portions of leaflets. Uredia are small and ostiolate, the conspicuous peridium ending in 4 to 6 knoblike spiny ostiolar cells. Urediospores are obovate or oblong-ellipsoid, 10μ to 18μ x 15μ to 26μ; the contents are bright yellow; walls are colorless, echinulate,

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FIGURE 17.—Late leaf rust on underside of raspberry leaflet.

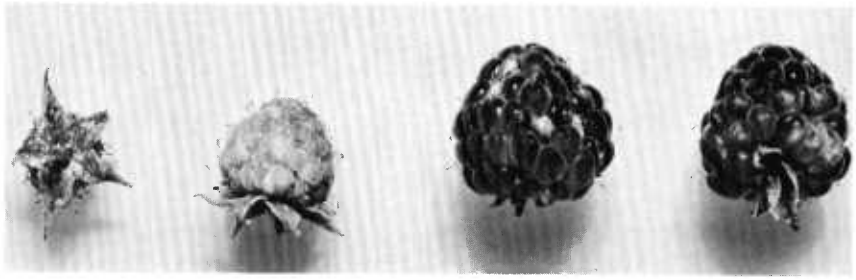

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FIGURE 18.—Late leaf rust on raspberry fruit.

1μ to 1.5μ thick. The small indehiscent telia are hypophyllous, brown, ellipsoid, smooth walled; vertically septate teliospores, 24μ to 28μ, are produced (*9*). Spermogonial and aecial stages on *Picea* spp. have been described and connected experimentally with the uredial and telial stages on *Rubus* including *R. strigosus* Michx. and × *R. neglectus* Peck (*89*). The white spruce (*Picea glauca* (Moench) Voss) (=*P. canadensis* (L.) B.S.P.) is an alternate host, but late leaf rust rarely occurs on spruce (*89*). In its absence the fungus apparently lives over winter on raspberry canes (*107*).

A closely related but physiologically distinct rust fungus, *Pucciniastrum arcticum* (Lagerh.) Tranz., also occurs on *Picea glauca* and northern *Rubus* species not including *R. strigosus* or × *R. neglectus* (*89*). *Pucciniastrum americanum* and *P. arcticum* will not cross-infect their respective *Rubus* hosts (*89*), but Dodge (*107*) considered the morphological variability in *P. americanum* to be great enough to include *P. arcticum* types, although he did not reduce the two species to synonomy.

Host resistance.—Among wild *Rubus* species, only *R. strigosus*, × *R. neglectus*, *R. leucodermis* Dougl., and *R. melanolasius* Focke are known to be susceptible to *Pucciniastrum americanum* (*9*). There are marked differences in susceptibility among red raspberry varieties. Latham, Ottawa, and Viking are susceptible; Chief and Newburgh slightly susceptible (*172*, *266*). Cultivated black raspberries and blackberries are resistant.

Control.—The disease is usually allowed to go uncontrolled. Use of management practices favoring good air movement around the plants and plowing under fallen rusted leaves may be desirable (*248*, *266*). Spraying may be done with ferbam (*266*). In northern Michigan eradication of white spruce near raspberry plantings has been suggested (*134*).

Leaf Spot [15]

Leaf spot of raspberries, although found in almost all sections of the United States where raspberries are grown, is most abundant and does the most damage in the southeastern quarter of the country.

[15] Caused by *Sphaerulina rubi* Demaree & M. S. Wilcox; imperfect stage is *Cylindrosporium rubi* Ell. & Morg.

The disease has also been erroneously called "Septoria leaf spot." The fungus (possibly fungi) causing raspberry leaf spot has been studied by Roark (*252*), Zeller (*353*), and Demaree and Wilcox (*100*).

Symptoms.—On new leaves greenish-black, circular to angular spots develop on the upper surfaces. As the leaves mature, the spots become whitish or gray and enlarge to 1 or 2 mm. in diameter, sometimes to 4 to 6 mm., but retain a well defined margin (fig. 19, *A*). The leaf lesions sometimes drop out, producing a shothole effect (*134*). Badly infected leaves drop off prematurely. Inconspicuous cane lesions, sometimes necrotic, also develop, particularly toward the bases of the canes (fig. 19, *B*).

Causal organism.—Perithecia are usually numerous, scattered or in groups, mostly hypophyllous, innate, erumpent, black, conical ostiolate-papillate, 88μ to 140μ x 86μ to 120μ. Paraphyses are lacking. Asci are fasciculate, sessile, clavate-cylindrical, curved or straight, 8-spored, 44.8μ to 70.0μ x 9.6μ to 15μ. The outer wall of the ascus is about 2μ thick, the inner wall membranous. Ascospores are hyaline, granular, cylindrical, usually curved, pointed at both ends, 4- to 8-celled (usually 4), 32.0μ to 57.6μ x 3.5μ to 5.8μ. Pycnidia are epiphyllous, subepidermal, 58μ to 80μ x 58μ to 121μ, lacking an ostiole. Pycnidial walls are thin, 1 to 3 cells thick. Conidia (pycnospores) are hyaline, elongate, obclavate, slightly curved to falcate, pointed at one end, 3- to 9-septate, 32μ to 86μ x 3.0μ to 4.8μ (in the region of greatest thickness) (*100*).

On raspberry, Demaree and Wilcox (*100*) regard *Rhabdospora rubi* Ell., *Septoria darrowi* Zeller, and *Septoria rubi* West. (the imperfect stage of *Mycosphaerella rubi* Roark) as synonymous with *Sphaerulina rubi*. However, Zeller (*353*) presents data showing marked differences in pathogenicity, leaf-spot-symptom production, and fungus morphology between collections of leaf-spot-infected raspberry leaves from Maryland and such leaves from Oregon, suggesting a lack of homogeneity in the fungi causing raspberry leaf spot. Workers agree, however, that the fungus or fungi that cause leaf spot on raspberries and those that cause leaf spot on erect and trailing blackberries do

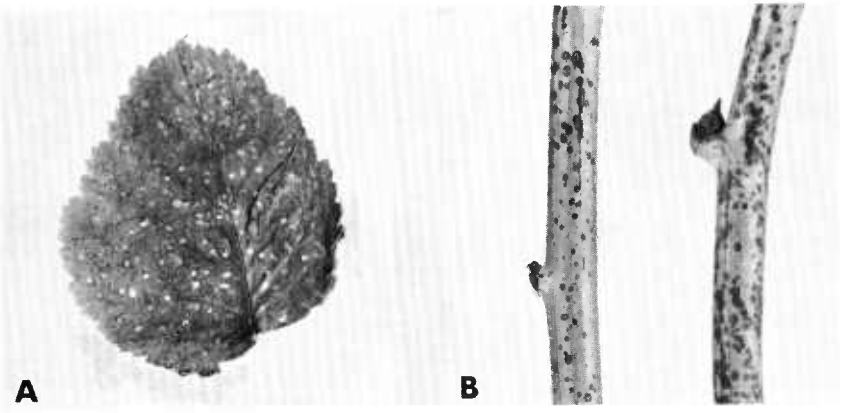

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Figure 19.—Leaf spot disease of red raspberry: *A*, Leaflet; *B*, stems.

not cross-infect under field conditions (*100*, *252*). Additional study is needed of this group of leaf-spotting fungi on *Rubus*.

In the Southern United States a leaf-spotting fungus appropriately named *Mycosphaerella confusa* Wolf having *Cercospora rubi* Sacc. as its imperfect stage also occurs on raspberries and blackberries (*337*).

Disease cycle.—*Sphaerulina rubi* overwinters in Maryland commonly both as the perithecial stage on dead leaves and as the winter pycnidial stage on leaves and canes. Only young expanding leaves and canes are susceptible to infection, and pycnidia develop on them during the summer, exuding spore masses, which disintegrate and are spread by rain or dew (*100*). Defoliation from heavy raspberry leaf-spot infection results in late summer and early fall, making the plants more liable to winter injury (*91*). Perithecia form in late fall on old leaves, and asci mature in the spring to release ascospores without forcible discharge. The pycnidia formed on canes are larger and thicker walled than those on leaves and do not form spores until they have overwintered. Waxy masses of pycnospores from cane lesions constitute an important source of inoculum in the early spring (*100*).

Host resistance.—The known host range of *Sphaerulina rubi* is limited to the genus *Rubus*. The susceptibility of wild American *Rubus* species in the subgenus *Idaeobatus* has not been well studied, but *R. strigosus* Michx. and *R. occidentalis* L. are susceptible. All Asiatic species of *Idaeobatus* tested and the red raspberry varieties Van Fleet and Dixie, with Asiatic *Rubus* parentage, were reported resistant in Maryland (*91*). Multiple factors for resistance are involved (*284*). Many other red raspberry varieties are susceptible in Maryland and elsewhere, including Chief, Latham, Newburgh, Taylor, Viking, and Washington; but Ranere (St. Regis) is grown in warm areas primarily because of its resistance (*182*). Several black raspberry varieties are reported susceptible, including Cumberland, New Logan, and Plum Farmer (*64*, *91*). *Eubatus* species of erect and trailing blackberries are resistant (*100*).

Control.—Management conditions that favor good air drainage and allow the plants to dry out rapidly after rain reduce the development of leaf spot (*134*). Pruning out old fruiting canes and dead wood is also valuable. The fungicidal spray schedule used to control anthracnose (pp. 25–26) is currently used in controlling raspberry leaf spot, which is not generally a severe problem except in the southern limits of raspberry production.

Orange Rust [16]

Orange rust is the most important of the several rust diseases attacking brambles. The fungus spreads throughout the roots, crown, and shoots of an infected plant and is perennial in the belowground parts. All varieties of black raspberries, but none of red, are affected. Some blackberries are susceptible. Orange rust is common in the northeastern quarter of the United States and occurs occasionally in the Pacific Northwest. Nonbramble plants are not attacked by this rust fungus.

[16] Caused by *Gymnoconia peckiana* (Howe) Trott. (=*G. interstitalis* (Schl.) Lag.).

The biology of the orange rust fungus, extensively studied by Dodge and others, was summarized by Dodge in 1923 (*109*).

Symptoms.—When the young plants of black raspberries received from a nursery are infected, the new shoots that develop are weak and spindly, lacking spines, and are very susceptible to powdery mildew (*Sphaerotheca humuli* (DC.) Burr.) (p. 39). Their leaves are small and yellowish. Within 2 or 3 weeks the undersides of leaves are covered with blisterlike aecial pustules from which reddish-orange aeciospores are shed (fig. 20, *B*). The young canes may appear to "grow out" of the rust toward the end of June, as the upper leaves then bear no rust. The canes are thoroughly infected, however, and will not blossom the next year. Most of the leaves show the pustules of orange rust each spring as long as the plant lives. The numerous, small, upright shoots of systemically infected plants are usually too weak to form rooted tips (fig. 20, *D*).

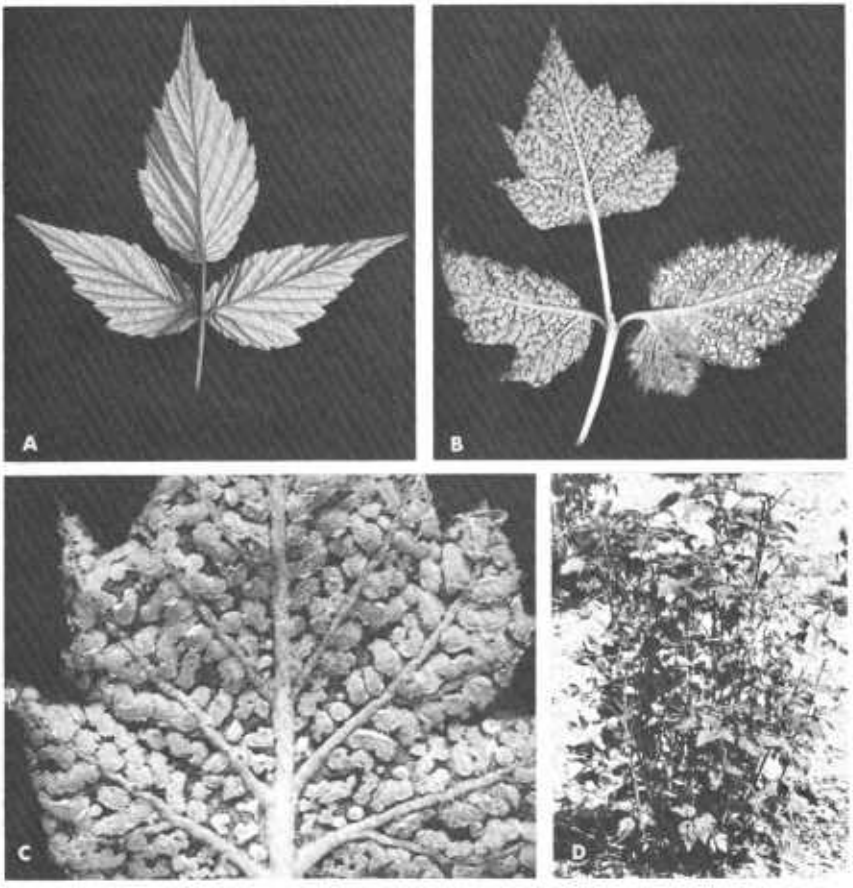

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FIGURE 20.—Orange rust of black raspberry: *A*, Healthy leaf; *B*, infected leaf showing aecial pustules; *C*, enlarged portion of infected leaflet; *D*, infected plant showing spindly, bushy growth.

Sometimes in old hills only a few of the canes show rusted leaves in the spring, whereas the other canes blossom and bear fruit normally. In such cases the fungus invaded the plant after it was set and has not yet reached all parts of the plant.

Causal organism.—*Gymnoconia peckiana* is an autoecious rust lacking the uredial stage. Small subcuticular spermogonia (pycnia), 100μ x 99μ, lacking peridium or paraphyses, are formed on leaflet upper surfaces and originate subepidermally (*164*). On blackberry a short-cycle form, *Kunkelia nitens* (Schw.) Arth., has also been described (*8*) and considered as a form of *G. peckiana* (*108*). (See p. 73.) Aecial pustules form on lower surfaces of black raspberry leaflets from perennial mycelium. Catenulate aeciospores are borne on a subepidermal hymenium. Aeciospores are orange colored in mass, globoid, 16μ to 24μ x 19μ to 30μ, finely verrucose with hyaline 1.5μ walls. Aecia have been formed in tissue cultures of systemically infected leaflets (*3*). *Gymnoconia peckiana* aeciospores usually germinate by germ tube; and true, dark-brown telia are produced on the underside of infected leaflets. The telia produce pedicellate, 2-celled, smooth-walled, ellipsoid brown teliospores, 18μ to 27μ x 32μ to 45μ, which form promycelia and sporidia (*9*). Both aeciospores and sporidia are forcibly discharged (*110*).

Disease cycle.—The orange aeciospores that fall upon leaves of raspberries in May or June infect only small areas of individual mature leaves. Penetration is direct by means of an appressorium and an infection peg. Mycelium is intercellular with haustoria penetrating parenchyma cells (*233*). In August and September small brown telial spots develop on the underside of infected leaflets. The teliospores borne in these brown spots produce sporidia that infect the buds on the cane tips just rooting. Mycelium overwinters in the infected host tissues. The orange aeciospore stage then develops in these rooting shoots the next spring, when the tips are starting to grow. Systemic infection through the cane to tips that are rooting is rare, because such canes are usually too weak to tip-root (*108*).

Buds or new shoots being formed at the crowns of healthy plants in late summer may become infected then. The fungus afterward grows down into the crown at the base of the affected shoot and enters newly formed roots. As a result, a few canes in the hill will show rust the following year. The possible role of overwintering teliospores producing basidiospores in the spring that could infect developing black raspberry buds has not been clarified.

Host resistance.—Satisfactory resistance is not known in commercial black raspberries. No authenticated instances of infection of red raspberry are known (*305*), but purple raspberry is reported susceptible (*9*). Physiologic races may exist in *Gymnoconia peckiana*. However, aeciospores from blackberry will infect black raspberry and vice versa (*108*).

Control.—Orange rust is most commonly spread in infected nursery stock. New plantings should obviously be started from rust-free stock. If the new shoots from tip plants show rusted leaves during the season they are set out, the tip plants were already infected when they were dug. Such plants are worthless and should be quickly dug up and burned before the orange rust pustules mature and discharge

spores. Inspection of dormant nursery stock will not reveal the presence of orange rust. In the spring when pustules are forming, but not open, is the time to check for the presence of orange rust and to rogue intensively. Growers who wish to sell orange-rust-free stock remove all infected plants before the rust pustules break open and discharge spores. Otherwise, many of the rooted tips would be infected by orange rust, even though they remained symptomless until the following year.

In case an old hill shows a cane or two whose leaves are rusted while other canes blossom normally, the part of the crown bearing the infected canes should be cut out the burned. It is sometimes possible in this way to save the plant (*108*). Infected wild black raspberries and blackberries near a black raspberry planting should be destroyed before the orange rust pustules on their leaves mature and release spores.

Some reduction in orange rust can be achieved by using management practices that favor good air circulation around the plants to let the foliage dry out rapidly. The orange rust spores require long periods of wetting before they germinate and infect plants. Fungicidal sprays used for control of anthracnose and other diseases (pp. 25–26) probably reduce the number of new infections of orange rust but should not be relied on to eliminate the disease.

Powdery Mildew [17]

Powdery mildew is usually a problem only on a few susceptible red, purple, and black raspberry varieties. The disease occurs throughout the raspberry-growing regions of the United States and varies greatly in severity from year to year. The most detailed study of the disease is by Peterson and Johnson (*237*). Downy mildew, caused by *Peronospora rubi* Rab., has been reported in Washington on black raspberry (*305*), and in Utah on red raspberry (*303*). In Utah it is a minor leaf disease in years of high rainfall (*303*).

Symptoms.—Leaves develop light-green blotches on the upper surfaces (fig. 21), matched on the lower sides by mealy white growth of the powdery mildew fungus. The mildew spots may appear to be watersoaked. Often leaves become mottled by *Sphaerotheca humuli* infection, but because fungus growth is sparse the leaves appear to be infected by mosaic. Shoot tips may be covered with the mealy growth and become long and spindly (rattailed), the leaves being dwarfed. The fruit itself is sometimes covered by the fungus. Powdery mildew can stunt plants when it is severe.

Causal organism.—*Sphaerotheca humuli* produces abundant, superficial mycelium and haustoria into host epidermal cells. Conidiophores bear catenulate, oval, unicellular conidia. Reddish-brown cleistothecia—Salmon (*257*) felt that they were true perithecia—are formed on host surfaces in the fall under circumstances not clearly understood. Cleistothecia are 58μ to 120μ in diameter with long, straight or twisted dark-brown appendages. A cleistothecium containing a single 8-spored ascus is elliptical to subglobose, 45μ to 90μ x 50μ to 72μ. The ascospores average 22μ x 15μ (*256*). Ascospores

[17] Caused by *Sphaerotheca humuli* (DC.) Burr.

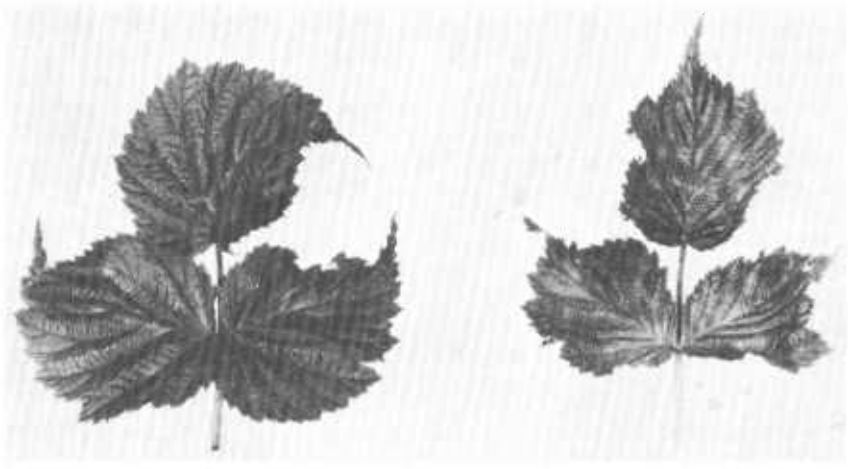

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FIGURE 21.—Powdery mildew on red raspberry leaves. Note the light discolored areas and the curled margins. (After Fulton, 1960.)

require a winter rest period in the cleistothecium in order to germinate (*257*). Physiologic races of *S. humuli* occur on hop, rose, strawberry, *Potentilla*, and other hosts, as well as on *Rubus* (*256*). The host ranges of the powdery mildews from these different hosts have not all been clearly established. The hyperparasite *Cicinnobolus cesatii* DBy. is very commonly found on old mildew mycelium; it may interfere with formation of the cleistothecial stage of *S. humuli* (*237*).

Disease cycle.—The fungus lives over the winter as mycelium in the buds on shoot tips, in Minnesota and probably in the Eastern United States (*237*). The histology of bud infection has not been reported.

The cleistothecial stage is not known on cultivated raspberries in the Eastern United States (*237*), but was reported to occur abundantly in California where overwintering cleistothecia produced ascospores to initiate spring infections (*204*). Overwintering mycelium was not found in buds on infected canes in California (*204*).

Repeated cycles of infection by air-borne conidia occur during the summer, but conidia are not viable for more than 21 days (*237*). Warm and cloudy weather with heavy dews or prolonged rainy periods are reported to favor infection in Michigan (*26*, *134*), but in New York mildew is reported to be prevalent in dry years (*266*).

Host resistance.—Most varieties of red, purple, and black raspberry are resistant to powdery mildew. As a group, red raspberries are least resistant. Loganberry (*332*) and erect and trailing blackberries (*25*) are generally resistant, although the trailing variety Lucretia is sometimes damaged by powdery mildew in Michigan (*345*). Latham, Ottawa, Puyallup, and Viking red raspberries; Cardinal purple raspberry (now a minor variety); and Black Hawk and Dundee black raspberries are susceptible (*25*, *134*, *172*, *237*, *261*, *266*).

Control.—Removal of late-formed mildewed suckers in the fall and cutting back fruiting canes to a horticulturally desirable height in the spring will help to reduce the sources of spring inoculum in an infected planting in the Eastern United States (*237*). Spacing the planting to allow good air drainage and maintaining narrow hedgerows or single plants is also helpful. The common fungicides used for anthracnose and spur blight control are ineffective for powdery mildew control. Sulfur dust is recommended in California (*62*) but has been phytotoxic elsewhere. In Oregon (*118*, *311*) and Washington (*145*), but not in Michigan (*128*), 2,4-dinitro-6- (2-octyl) phenyl crotonate, is recommended for the control of powdery mildew on raspberries as thorough weekly sprays from petal opening until petal fall. Care must be taken not to exceed recommended dosages and, to avoid burning the foliage, not to spray in hot weather (*311*). The material cannot safely be applied nearer than 7 days to harvest, and its residue tolerance is zero (*304*). Dormant application of lime-sulfur aids in powdery mildew control in Oregon (*118*) but not in Michigan (*345*) or Minnesota (*237*). Where powdery mildew is a persistent and serious problem on susceptible varieties in the Eastern United States, substitute varieties should be planted (*266*).

Root Rots

In addition to Verticillium wilt (p. 46) there are several other fungi that attack roots of bramble crops, causing root rots that weaken or kill the plants. *Rubus* root rots have yet to be intensively studied, and the etiology of some important root rots is still poorly understood. The fungi associated with the common *Rubus* root rots have been studied (*27*, *68*, *147*, *241*, *303*, *314*, *319*, *333*). Commonly isolated were *Cylindrocarpon radicicola* (McAlp.) Wr., *Fusarium* sp., *Pythium* sp., *Coniothyrium fuckelii* Sacc. (see footnote 7, p. 26), *Rhizoctonia* sp., and *Phoma* sp. The primary pathogens, the sequence of infection, and the factors influencing the development of *Rubus* root rots require much intensive study. Likewise does the role of root rots in various imperfectly understood disorders, such as those known as root degeneration, heavy soil disease, wet feet, and winter injury.

Root rot of certain red raspberry varieties on heavy soils in the Pacific Northwest is regarded as a serious disease of the crop (*112*, *262*). The etiology of the disease is unknown. While most varieties are susceptible (*112*, *262*), Fairview, Newburgh, and Sumner appear to have some resistance.

The role of *Phycomycetes* in *Rubus* root rots has received attention. Unidentified *Phycomycetes* were reported in 1940 to attack raspberry feeder roots in Oregon (*354*) and British Columbia (*27*). McKeen (*212*) established the existence in British Columbia of an isolate of *Phytophthora fragariae* Hickman, the incitant of strawberry red stele root rot, that was capable of causing a root rot of Loganberry and Cascade varieties of blackberry. Converse and Scott (*82*) were unable to repeat this work in Maryland using an isolate from McKeen that had been in culture for 4 years. McKeen (*211*, *212*) suggested that root degeneration of raspberry was probably caused by a race of *P. fragariae* of unknown relationship to the Loganberry race C–1, but no supporting data were presented. Jarvis and Montgomerie (*181*)

have briefly reported a root rot of raspberry in waterlogged fields in Scotland apparently caused by *P. cactorum* (Leb. & Cohn) Schroet., which may be related to a *Phytophthora* species associated with raspberry "die-back" in Scotland by Waterston (*319*).

Armillaria, or mushroom, root rot, caused by *Armillaria mellea* Vahl ex Fr., is reported from Texas and the Pacific States on all cultivated brambles (*305*). The disease is not generally serious but often occurs on land recently cleared of wild oaks (*315*). Affected plants wilt, temporarily recover, then collapse and die suddenly. The disease spreads out year after year in widening circles in the field from points of infection. Strands of mycelium (rhizomorphs) move through the soil, and the fungus invades the roots of adjacent plants. Thick sheets of white fungus hyphae having a mushroomlike odor are seen under the bark of infected plants (*11*). Sporophores of *A. mellea*, commonly called the oak root fungus, are seldom seen. The spread of Armillaria root rot can usually be prevented by removing a single ring of plants around the infected area, as well as by digging out infected plants and as many infected roots as possible. Carbon disulfide fumigation will sterilize infested soil (*62*). *Collybia dryophila* Fr. causes a similar disease of Lucretia trailing blackberry in North Carolina (*240*).

Spur Blight [18]

Spur blight is a serious disease of red raspberry in the Northern States, seldom found on other brambles. Defoliation, killing of buds, and predisposition to winter damage resulting from infection by the spur blight fungus have accounted for 25-percent losses in some areas (*192*). In one Michigan test, uncontrolled spur blight was found in 60 percent of Latham buds (*135*). The biology of the disease and the causal organism were extensively studied by Koch (*201*). Bolton and Julien (*32*), Julien and Bolton (*192*), have studied variation in the causal organism.

Symptoms.—When canes are 9 to 15 inches high, small brown or purple spots begin to appear mainly on the epidermis at the nodes on the lower portion of the stems. To some extent the spots are found on petioles and on leaflets where brown wedge-shaped lesions develop (fig. 22). In the typical infection, the region around the buds turns brown (fig. 23); the buds shrivel and may die. If the buds live over winter, they produce only small yellowish leaves but no spur. Buds near the ground are most likely to be attacked; infected fruiting canes often have few fruiting spurs left within 18 inches of the ground. New lesions can occur on healthy fruiting canes, but the fungus usually does not attack flowers or fruit. Leaf blades fall off, leaving the petioles attached to the cane. As diseased canes mature, they dry out and may crack, and lesions turn gray and may enlarge to cover much of the cane.

Causal organism.—Perithecia of *Didymella applanata* form readily in the fall in grayish lesions on infected canes. Perithecia are distributed at random on the lesion rather than in the concentric pattern

[18] Caused by *Didymella applanata* (Niessl) Sacc.; imperfect stage is a *Phoma* sp.

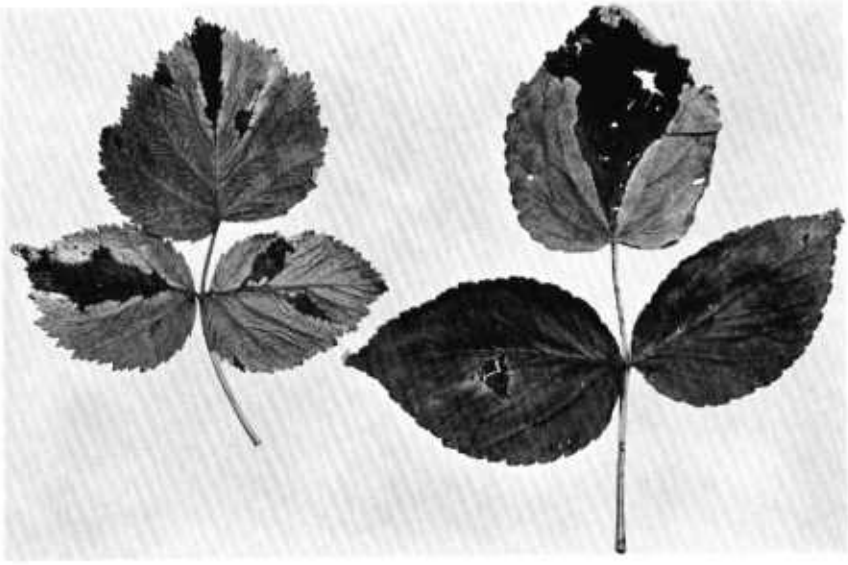

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FIGURE 22.—Spur-blight-infected red raspberry leaves. Note the dark lesions extending out to the leaflet margins. (After Fulton, 1960.)

displayed by perithecia of the cane blight fungus (*Melanomma coniothyrium* (Fckl.) L. Holm), which frequently is also present on infected overwintering canes (p. 26). Perithecia are black, commonly gregarious, flattened to subglobose, averaging 195μ in diameter; they are subepidermal, becoming superficial when the epidermis is lost. Asci are cylindrical and fasciculate, 60μ to 70μ x 10μ to 12μ. Ascospores are matured and forcibly discharged from overwintered perithecia during rainy periods from May through June (*201*). Ascospores are usually biseriate, hyaline, obovate to oblong, uniseptate, one cell being larger than the other, 16μ x 5.6μ. Pycnidia (of an unnamed *Phoma*) are black, separate, smooth, averaging 208μ x 187μ, innate-erumpent, borne free in the substratum on young tissue, often intermingled with perithecia in old lesions. Conidiophores are simple. Spores are hyaline to light green, mostly 2-guttulate, elliptical to oval, 7.1μ x 2.9μ. Frequently release of conidia occurs simultaneously with ascospore discharge (*201*). Survival of the fungus in bramble debris has not been studied. Pycnidia containing viable conidia have been produced in pure culture (*201*), and perithecia are formed in culture on 1.5 percent water agar (*192*). The nature of sexuality in *D. applanata* has not been reported.

Disease cycle.—Ascospores discharged from perithecia on infected fruiting canes in late spring may infect young shoots or leaves. Discharge occurs only in rainy periods (*201*). Conidia may also begin infections. Moist conditions are required for germination and infection, and germ tubes can penetrate wounded or unwounded host epidermis (*201*). Moisture retention in the region of buds makes them favorable infection courts for *Didymella applanata* (*186*). Paren-

BN–22642

FIGURE 23.—Primocanes of red raspberry showing spur blight infections (darkened areas). (After Fulton, 1960.)

chymatous host tissues are usually invaded intracellularly. Intact unbroken layers of cork cells serve as a barrier to the fungus (*201*). Vascular invasion seems not to occur. Infection of leaf or stem leads to browning and death of tissue. Infection of buds appears to progress from the bud tip down rather than from the infected cane into the bud (*201*). Petiole infection often spreads down into the node (*346*).

Pycnidia formed in infected tissues may release spores during the summer that can infect young tissues. Perithecia and pycnidia then develop in cane lesions over winter.

In Europe the raspberry cane midge (*Thomasiniana theobaldi* Barnes) produces wounds in young red raspberry canes, which frequently are invaded by the spur blight fungus, the cane blight fungus, and *Fusarium culmorum* (W. G. Sm.) Sacc. (*238*). The raspberry cane midge is not known to occur in North America.[19]

Host resistance.—The fungus causing spur blight attacks only brambles. It is common on wild and cultivated red raspberries, particularly on older plantings, and on some other species in the *Rubus* subgenus *Idaeobatus;* it is, however, less common on black and purple raspberries, and does not infect many members of the subgenus *Eubatus* other than Loganberry.

There are differences in varietal susceptibility to spur blight among red raspberries. Seedlings that are hairy, spinefree, nonpigmented, waxfree, or moderately densely waxed are considerably more resistant to spur blight than seedlings not having these characters (*186*). Finnish data (*143*) indicate a highly significant correlation ($r = +0.615$) between susceptibility to spur blight and susceptibility to colonization by the spider mites *Phyllocoptes gracilis* (Nal.) (=*Eriophyes gracilis* (Nal.)) and *Tetranychus urticae* Koch (=*T. altheae* Hanst.), both of which occur in the United States.[20]

Varieties resistant in one area are not always so resistant in another. At least two races of *Didymella applanata* are known to exist in eastern Canada, differing in their ability to attack the variety Newburgh (*32*). Physiologic specialization has not been reported in the United States.

No red raspberries are known to be immune from spur blight, but there are differences in susceptibility among varieties, Marcy and Washington being moderately resistant (*64*, *145*, *289*), Taylor and Willamette being very susceptible (*145*, *201*, *289*). Like Newburgh, the response of Latham is variable (*46*, *289*). Breeding projects for spur blight resistance are in progress (*186*, *192*).

Control.—The cultural practices and spray schedules recommended for anthracnose control (pp. 25–26) have been valuable for spur blight control. When spur blight is the main problem, the dormant spray (preferably DNOC) should be delayed until the late green-tip stage of bud breaking (about 7 days later than for anthracnose); the first protectant spray (such as ferbam) should be applied when the canes are 9 to 10 inches high, followed by another protectant spray in about 2 weeks (*289*).

Fields severely infected with spur blight cannot be brought into good fruiting condition that same season by fungicidal sprays, since

[19] Foote, R. H. Personal communication, 1961.
[20] Baker, E. W. Personal communication, 1964.

the fungus has already damaged the fruiting canes. A new planting generally requires several years to become severely infected with spur blight.

Because the fungus does not live over in the roots, a badly infected field can be renovated by cutting off and burning all canes before spring ascospore discharge and by spraying the rows with lime-sulfur or DNOC. New shoots relatively free from spur blight should then develop. Durham variety of red raspberry was selected specifically for the fall production of fruit under this system (*343*).

Verticillium Wilt [21]

Verticillium wilt of raspberries, sometimes called bluestem and blue stripe wilt, is serious and destructive to raspberries in the northern half of the United States and along the Pacific coast. It is a soil-borne fungus disease that is particularly damaging to and widespread on black raspberries. In Oregon, Verticillium wilt was detected in 34 percent of the black raspberry fields sampled in one survey (*242*). The early literature on Verticillium wilt, which has an extensive host range across many plant families, was reviewed by Rudolph in 1931 (*254*). Buxton (*48*) has briefly reviewed the wilt diseases in general. The literature specifically on Verticillium wilt of brambles was reviewed by Wilhelm and Thomas (*329*), Fulton (*130*), and Pratt (*242*). Verticillium wilt on brambles in England was studied by Harris (*146*).

Symptoms.—On black and purple raspberry new infections of Verticillium wilt cause leaves of new canes to turn pale in midsummer. Plants may appear to recover during the cool fall weather, but in the following spring leaves on infected fruiting canes, beginning at the bottom and moving upward, may turn yellow, then wilt and die. Infected canes are stunted and often turn entirely blue or may be blue on one side, before they wither and die (fig. 24). The bluish tinge imparted to the cane accounts for the name "bluestem." Infected vessels in the xylem generally have a red discoloration (*130*). The plants usually die in 1 to 3 years (*25*).

All symptoms on red raspberry are generally less severe than on black raspberry. Leaf symptoms are similar. Often leaflets fall before the petioles drop. A tuft of leaves may remain at the tip on infected canes. Cane discoloration is not so evident as it is on black raspberry. Red raspberry plants may survive for years, but the amount of suckering is reduced (*130*, *254*).

Verticillium wilt may infect only part of a raspberry plant if the fungus has not invaded all the roots, so part of the plant may be dying while the rest appears to be healthy.

Field symptoms are not always a reliable indication of the presence or absence of Verticillium wilt. Other root rots, insect feeding, wind damage, and high water content of the soil can produce similar symptoms, and in some cases *Verticillium albo-atrum* can be recovered from symptomless plants (*242*). Use of a solanaceous trap crop to identify *V. albo-atrum* in suspect fields has been suggested (*242*, *326*).

[21] Caused by *Verticillium albo-atrum* Reinke & Berth.

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FIGURE 24.—Verticillium wilt of black raspberry plant.
(After Fulton, 1960.)

Causal organism.—There is disagreement in the literature about the identity of the fungus or fungi causing Verticillium wilt in general and Verticillium wilt of *Rubus* in particular. Both *Verticillium albo-atrum* Reinke & Berth. and *V. dahliae* Kleb. are reported as causes of Verticillium wilt in *Rubus* (*47*, *254*). The difficulty appears to lie in the question of the validity of the two species, as some maintain (*174*, *195*, *209*, *229*, *253*), or the reduction of *V. dahliae* to synonomy under *V. albo-atrum*, which has priority, as preferred by others (*16*, *66*, *116*, *322*). A differential medium has been developed to separate the two species (*295*). Workers who are not in agreement with regard to the existence of distinct species (*16*, *116*, *209*) recognize the existence of a group of isolates of *V. albo-atrum* having dark hyphae that is more sensitive to 35° C. in pure culture than the group of microsclerotial isolates (*V. dahliae*). On the other hand, Wilhelm and Nelson (*228*, *322*) found that temperature played a major role in the production of microsclerotia by colonies of *V. albo-atrum* on agar; they noted that the thermal death point of hyphae and conidia of *V. albo-atrum* was 47° C. Fulton (*130*) found that monoconidial isolates from raspberry could be divided into four biotypes differing in colony morphology but all pathogenic to raspberry.

Berkeley and Jackson (*29*) reported the occurrence of a new species, *Verticillium ovatum* Berkeley & Jackson, on red raspberry in Ontario, which Rudolph (*254*) designated as *V. albo-atrum* Reinke & Berth. var. *ovatum* Berkeley & Jackson.

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In the present treatment the fungus causing Verticillium wilt of brambles will be considered to be *Verticillium albo-atrum* in keeping with the viewpoint of several American authors on the subject (*130*, *254*, *329*, *352*); also, the description will be based upon that by Carpenter (*66*). For a review of *V. dahliae* as the etiologic agent of Verticillium wilt in brambles the reader is referred to the excellent treatment by Butler and Jones (*47*).

Conidiophores are 100μ to 300μ or more in length, developing whorls of branches. There are 1 to 8 primary whorls per conidiophore, usually 30μ to 90μ apart, sometimes bearing secondary whorls with straight or slightly bowed branches 13μ to 38μ in length. Conidia are borne singly on tips of sterigmata on the branches, but may occasionally collect into heads. Conidia are ellipsoidal, unicellular, 4.0μ to 11μ x 1.7μ to 4.2μ. The mycelium is septate and hyaline, turning brown with age. Hyphae may become changed into swollen chlamydosporelike knotted masses that aggregate into microsclerotia, sometimes called pseudosclerotia (*66*).

Disease cycle.—Much of the following information is drawn from studies of other crops to supplement the more limited amount of information available concerning Verticillium wilt of brambles. The fungus hyphae enter root hairs or penetrate root cortex directly and make their way to xylem vessels. Movement through the host is accomplished by growth of hyphae through the vessels and by movement of conidia in the transpiration stream (*116*). Upon the death of infected parts the fungus is returned to the soil, where it maintains itself in competition with the soil micro-organisms (*116*). The question of whether it is the microsclerotia themselves or conidia and hyphal fragments associated with microsclerotia that germinate and perpetuate the fungus in the soil has been a matter for much recent investigation and disagreement (*116*, *208*, *224*, *244*, *258*, *259*, *326*). The survival and germination of microsclerotia specifically from infected raspberry have not been studied, with the exception noted below (*352*).

Verticillium albo-atrum occurs primarily in the top 12 inches of soil but has been recovered down to 36 inches (*323*). The organism will survive at least 14 years in the soil in California in the absence of a known host, but at a low inoculum potential (*326*). Microsclerotia will not survive prolonged (6 weeks) flooding (*218*). *Verticillium albo-atrum* should be considered a soil invader, not a soil inhabitant (*116*, *324*).

Zeller in Oregon (*352*) found that *Verticillium albo-atrum* could be recovered by direct plating from infected raspberry canes buried in the soil up to 16 months. He was able to show that the organism moved through soils to cause new infections in raspberry plantings. Bioassays of inoculum potential rather than direct plating were used by Wilhelm (*326*, *327*) to demonstrate long-term survival in the soil. More recently a selective ethanol-agar medium has been devised for direct plating of *Verticillium* from the soil (*225*); this medium has demonstrated viability of *Verticillium* in soils planted to nonhosts (*215*).

The influence of soil type on the incidence of the disease in general was found to be negligible by Wilhelm (*323*), but there are reports that heavy soils have increased wilt incidence (*25*).

The role of conidia in dispersal of the fungus from plant to plant has not been extensively studied. Conidia are rather delicate and are adapted to dispersal by rain and insects (*325*). Conidia can form microsclerotia by anastomoses of germ tubes, a fact that may be of significance in the survival of the fungus (*325*).

The severity of the disease depends upon the inoculum potential in the soil, which in turn depends upon crop history (*228*, *323*). Degree of severity in raspberries depends also on the presence of a strain of *Verticillium albo-atrum* that is capable of causing Verticillium wilt of raspberries. Plantings of raspberries are generally most susceptible during the first few years of growth (*248*, *321*). In addition to its survival in soil, *V. albo-atrum* may sometimes occur in raspberry nursery stock without symptoms (*25*, *271*).

Zeller (*352*) and Bennett (*25*) found that the severity of Verticillium wilt in black raspberries is related to the coldness of the previous winter. Cold wet spring weather leads to higher incidence of Verticillium wilt on raspberries in Ontario (*248*). Slate, Braun, and Mundinger (*266*) feel that infected brambles in New York in times of high summer temperature may outgrow Verticillium wilt. Various other workers have reported that hot weather increases disease damage. A cool cloudy spring followed by hot summer weather caused Verticillium wilt of blackberries in California to be more severe (*329*). The fact that symptoms become acute in midsummer could well result from the combination of damage to vascular tissues by the fungus and the normal moisture stress of hot dry weather. Fulton (*130*) found Verticillium wilt severity in Michigan raspberries to be related to high summer temperatures at the time of symptom expression and suggested that differences in temperature tolerance of the isolates studied by various workers could account for the divergent results reported, a viewpoint that Butler and Jones (*47*) share at the species level.

The physiology of symptom production by *Verticillium albo-atrum* has received much attention recently. Interference with the normal water relations of the host by plugging of vessels with hyphae and with tyloses induced by the presence of the fungus appears to be central in the development of Verticillium wilt symptoms regardless of host (*292*). That toxins elaborated by *V. albo-atrum* are involved in the production of wilt symptoms in field-grown raspberry is suggested by the studies of Fulton (*129*), who demonstrated the ability of pure culture filtrates of *V. albo-atrum* to cause wilting and vessel discoloration in raspberry. Work with toxins in other hosts suggests that toxins may be involved in the wilting of plants (*214*, *300*) and in production of leaf symptoms (*294*). Indole acetic acid-like activity is increased in host tissues infected by *V. albo-atrum*, and water loss in leaves is due to resulting changes in membrane permeability according to Pegg and Selman (*236*) ; but the symptoms of water shortage are regarded by others to be due not to IAA-induced changes in cell permeability but rather to vessel blockage (*263*, *300*).

Host resistance.—The host range of *Verticillium albo-atrum* is very wide (*254*). Within the species, however, numerous physiologic races have been reported, each with its own limited host range. The possible occurrence of different races in various areas (*130*) makes it

difficult to predict what group of hosts will be associated with Verticillium wilt of raspberry in a given area.

The host range studies compiled in tables 2 and 3 are not presented as being exhaustive of the literature but to support the general conclusion drawn by Van den Ende (*116*) that the host range of *Verticillium albo-atrum* strains is rather broad. Experimental susceptibility of a crop does not necessarily mean that it will serve as an important

TABLE 2.—*Host sources of* Verticillium albo-atrum *pathogenic to brambles*

Host:	*Reference:*
Raspberry	*130, 254, 352.*
Apricot	*254.*
Black nightshade (*Solanum nigrum* L.)	*329.*
Cotton	*329.*
Groundsel (*Senecio vulgaris* L.)	*315.*
Peach	*118, 254.*
Potato	*329, 352.*
Rough pigweed (*Amaranthus retroflexus* L.)	*315.*
Solanaceae	*118, 266, 315.*
Strawberry	*242.*
Tomato	*254, 329.*
Most isolates tested	*242.*

TABLE 3.—*Hosts successfully infected by* Verticillium albo-atrum *from brambles*

Host:	*Reference:*
Raspberry	*130, 254, 352.*
Apricot	*254.*
Dandelion (*Taraxacum officinale* Weber)	*242, 254.*
Eschscholtzia californica Cham	*254.*
Lambsquarters (*Chenopodium album* L.)	*242.*
Medicago hispida Gaertn	*254.*
Mint [1]	*169, 242.*
Myrobalan plum	*254.*
Rough pigweed (*Amaranthus retroflexus* L.)	*242.*
Senecio vulgaris L	*242, 254.*
Solanaceae (a few species)	*130, 242.*
Strawberry	*16, 242.*
Tomato	*242, 254, 271.*

[1] Mint also *not* a host (*229*).

reservoir of inoculum in the field for subsequent raspberry plantings. For example, Pratt found strawberry isolates of *V. albo-atrum* to be pathogenic to black raspberry but failed to find Verticillium wilt in Oregon black raspberry fields planted after strawberries (*242*).

Repeated passage through a nonhost increased the virulence of one *Verticillium albo-atrum* isolate to the new host (*116*), suggesting a plasticity in host-range limitations for the species.

Field evidence on many crops indicates that avoidance of hosts susceptible to *Verticillium albo-atrum* lowers the inoculum potential of the fungus in the soil. However, long-term persistence at a low inoculum potential either on nonhost roots or as microsclerotia in the soil seems to occur in *V. albo-atrum*. The possibility of such sources of Verticillium wilt of raspberry should be established by direct study.

The evidence that some potato strains of *Verticillium albo-atrum* may exist in the soil for many years in symptomless condition on roots of nonhosts is presented by Martinson and Horner (*215*), but this was not found to be the case for tomato strains (*326*). Raspberry has been found to harbor the fungus in symptomless condition (*25*, *242*, *271*).

The nature of host resistance to Verticillium wilt has been extensively studied, particularly in the hop plant. In wilt-tolerant species of hop, invading hyphae may be sheathed by deposits from host cells. The endodermis of tolerant varieties is more heavily suberized than the endodermis of susceptible varieties (*113*, *293*).

Varietal resistance in raspberries is not adequate for control of the disease. All black and purple raspberries tested are very susceptible to Verticillium wilt (*271*). Among red raspberries Chief, Ranere (St. Regis), and Viking are mentioned as susceptible (*29*, *315*). Latham was susceptible in Oregon (*315*) but was somewhat more resistant than other varieties in Michigan (*130*). Cuthbert was very resistant in Oregon (*315*) but susceptible elsewhere (*25*, *29*), indicating the possible existence of physiologic strains of the fungus.

Control.—The use of clean nursery stock, avoidance of infested land, rotation and fumigation to disinfest the land when needed, and roguing of plants that become infected are the major control measures used for Verticillium wilt of raspberries. Foliage applications of fungicides are ineffective in control. In addition, as has already been noted, satisfactory varietal resistance in commercial raspberries does not exist.

It seems quite likely that the pruning and trimming operations in a raspberry planting or packing shed might transmit the disease. The experimental use of budding knives infected with *Verticillium albo-atrum* conidia resulted in high incidence of Verticillium wilt on roses (*246*). Only symptomless raspberry stock from fields known to be free from Verticillium wilt should be offered for sale, although Pratt (*242*) failed to recover *V. albo-atrum* from black raspberry tips that had rooted in infested soil.

The use of chloropicrin to fumigate *Verticillium*-infested soils has been brought to the commercial level in California through the work of Wilhelm and associates (*328*). Excellent control of Verticillium wilt of brambles on a field basis is obtained in California by a proper application of chloropicrin to infested land (*62*), but this is a very expensive operation.

Roguing a new planting that does not have more than about 5 percent infected plants is recommended as a means of preventing spread of the disease in the field (*315*). When infected plants are dug up, care should be taken to carry the plants and the soil immediately around them out of the field for discarding. Three- to four-year rotations into nonsusceptible crops have been recommended for control in Canada (*26*) but are not effective in California (*326*).

Yellow Rust [22]

This is generally a minor red raspberry leaf and cane rust disease, of worldwide distribution, locally and sporadically severe on certain susceptible varieties in the Pacific Northwest. It is also known as western yellow rust and cane rust. The life cycle of the fungus was studied by Zeller (*350*) and Zeller and Lund (*357*).

Symptoms.—Sparse to numerous orange-yellow aecia, less than 1 mm. in diameter, on upper surfaces of leaves are the first signs of the fungus noticed in the spring. In June orange to pale yellow uredia develop, 0.1 to 0.2 mm. in diameter on the undersides of leaves and several millimeters in diameter on the canes (fig. 25). Fruit infection has been reported (*287*). Defoliation may result from heavy leaf rust infection. The uredial pustules darken when black telia develop in them, from mid-July through late winter. Overwintering cane lesions become deep and cankerous, and infected fruiting canes may break or dry up the following summer. This is the most serious damage caused by the yellow rust fungus and the only injury, so caused, that can be related to a subsequent loss in yield (*308*). Like late leaf rust, caused by *Pucciniastrum americanum* (p. 32), yellow rust is not systemic in red raspberry. The production in yellow rust of orange-yellow aecia only on the upper surfaces of leaves is a simple character for distinguishing the two rusts.

Causal organism.—Spermogonia of *Phragmidium rubi-idaei* are epiphyllous, inconspicuous, few, surrounded by aecia. Aecia are epiphyllous in small annular groups, pulvinate, orange yellow when fresh with few, inconspicuous, peripheral paraphyses. The aecial walls are evenly thin; aeciospores are globoid or broadly ellipsoid, 14μ to 20μ x 16μ to 24μ, the walls pale yellow; sparsely echinulate-verrucose, 1.5μ to 2.5μ thick. Uredia are hypophyllous, incurved, with numerous peripheral paraphyses. Urediospores are broadly ellipsoid, 15μ to 18μ x 18μ to 23μ, the walls pale yellow, strongly echinulate, about 1.5μ thick, the pores obscure. Telia are hypophyllous, arising in old uredia, blackish. Teliospores are cylindrical, 26μ to 30μ x 80μ to 120μ, 6- to 10-celled; walls dark chocolate brown, 6μ to 7μ thick, coarsely verrucose with a hyaline apiculus 3μ to 13μ long. Pedicels are colorless except near the spores, swelling in water to become lanceolate in the lower part (*9*). At least two physiologic races of *P. rubi-idaei* are known to occur in Washington (*188*).

Disease cycle.—Teliospores overwinter and produce promycelia with sporidia in the spring. Teliospores require several months of winter weather to become ready for germination; in the presence of host tissues ripened spores are stimulated to germinate. Sporidial germ tubes penetrate leaf surfaces directly and give rise to uninucleate mycelium from which spermogonia and aecia are developed. Emerging young shoots can be infected at the ground level and develop numerous spermogonia and sori (*227*). The first binucleate cells are in the erect hyphae. Germinated aeciospores can initiate new infections on leaves by stomatal penetration. Uredia are formed on leaves and canes and release urediospores that also infect through stomata

[22] Caused by *Phragmidium rubi-idaei* (DC.) Karst.

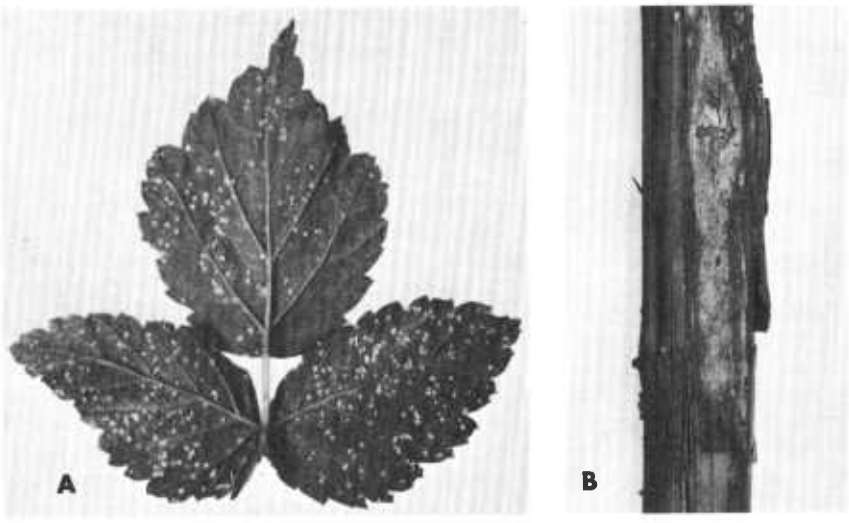

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FIGURE 25.—Yellow rust on red raspberry: *A*, Infected leaf; *B*, infected cane. (Courtesy of Washington Agricultural Experiment Station.)

(*357*). Cane lesions near cane bases enlarge, become cankerous, and are frequently invaded by the cane blight organism *Melanomma coniothyrium* (p. 26) (*349*, *351*). Telia formed in old uredia overwinter the fungus. Disease development is favored by cool, humid weather. Late spring rains favor a severe disease outbreak (*350*). The fungus requires moisture to be present on the infection court for several hours in order to invade the host (*357*).

Host resistance.—Only certain *Rubus* species are known to be susceptible: *R. arizonicus* (Greene) Rydb., *R. leucodermis* Dougl., *R. melanolasius* Focke, × *R. neglectus* Peck, *R. occidentalis* L., and *R. strigosus* Michx. (*9*). The brambles *R. parviflorus* Nutt. and *R. spectabilis* Pursh were immune when experimentally inoculated (*357*), but the latter was earlier reported very susceptible (*350*). Two other rusts, *Phragmidium rubi-odorati* Diet. and *P. occidentale* Arth., infect *R. odoratus* L. and *R. parviflorus;* their host ranges are not otherwise known (*9*). *Phragmidium violaceum* (C. F. Schultz) Wint. has been reported to cause fruit and leaf rust on blackberry in Great Britain (*147*), but it is not known in the United States (*305*).

The red raspberry varieties Cuthbert, Ranere (St. Regis), and Washington are susceptible to yellow rust (*188*, *357*), but Latham and Chief are highly resistant (*357*) and Puyallup is reported to be immune (*261*). Some cultivated purple and black raspberries were found to be somewhat susceptible under experimental conditions, but are not economic hosts (*357*).

Control.—Use of resistant red raspberry varieties in the few areas where yellow rust is a problem is probably the best control. Where susceptible varieties are so severely attacked that fruiting canes are damaged, control by plowing under old leaves and canes in early

spring (*350*) and use of lime-sulfur or other suitable fungicide at the green-tip stage but not later (*308*) will aid in control of the disease.

Bacterial Diseases

Cane Gall [23]

This disease is caused by a bacterium similar to that causing crown gall. (See p. 56.) The disease occurs widely on the fruiting canes of black and purple raspberries. Boysenberry and Himalaya blackberry are also susceptible (*161*, *210*, *310*) and red raspberry experimentally (*161*). Cane gall was described by Banfield (*12*), and a separate species was erected for the causal organism by Hildebrand (*161*).

Symptoms.—Whitish warts or ridges arise on the canes and darken as the canes enlarge; they cause the canes to split open (fig. 26). Such canes dry out, and the berries produced on them are small and seedy. The swellings caused by the *crown gall* bacterium are rarely seen above ground.

Causal organism.—Bacteria causing cane gall are Gram-positive rods 0.6μ x 1.7μ, occurring singly, in pairs, or in short chains. They are motile by means of 1 to 4 flagella. On agar *Agrobacterium rubi* colonies are filiform, white to creamy white with a butyrous consistency, later becoming leathery. Nitrite is not produced from nitrate by *A. rubi*, whereas there is a slight nitrite production from nitrate by the bacterium *A. tumefaciens* producing crown gall (p. 56). Unlike that bacterium, *A. rubi* cannot utilize asparagine as a sole source of carbon (*40*).

Hildebrand (*162*) described a new strain of *Agrobacterium rubi* from Boysenberry that would also infect *Kalanchoë* sp. experimentally. The experimental host range of *A. rubi* was further extended to *Vicia* sp. and *Helianthus* sp. (*74*) and to tomato (*196*).

A comparison of several isolates of *Agrobacterium rubi* and *A. tumefaciens* led McKeen (*210*) to conclude that although real differences exist in nature in host range and symptom production between these two species, intergrades between them occur.

Disease cycle.—Like crown gall bacteria (p. 58), the cane gall bacteria infect brambles through wounds. Bacteria survive in the soil in the absence of bramble plants for at least 6 months (*161*), and in Oregon from 5 to 6 years (*118*). The exact method of infection is not known. Authorities differ as to whether the bacteria spread from old galls to insect wounds (*161*) and pruning wounds (*161*, *210*, *310*), or whether they move within the plant (*161*, *210*). Winter injury promotes infection (*210*, *310*); McKeen (*210*) believes that this occurs through stimulation of bacteria that are already systemic in the canes.

Control.—Since cane gall is more readily seen than crown gall, plants with cane galls may be more easily rogued. Choice of clean nursery stock and uninfested land is important in cane gall control. Many of the details of control of crown gall (p. 58) also apply to cane gall control.

[23] Caused by *Agrobacterium rubi* (Hildebrand) Starr & Weiss.

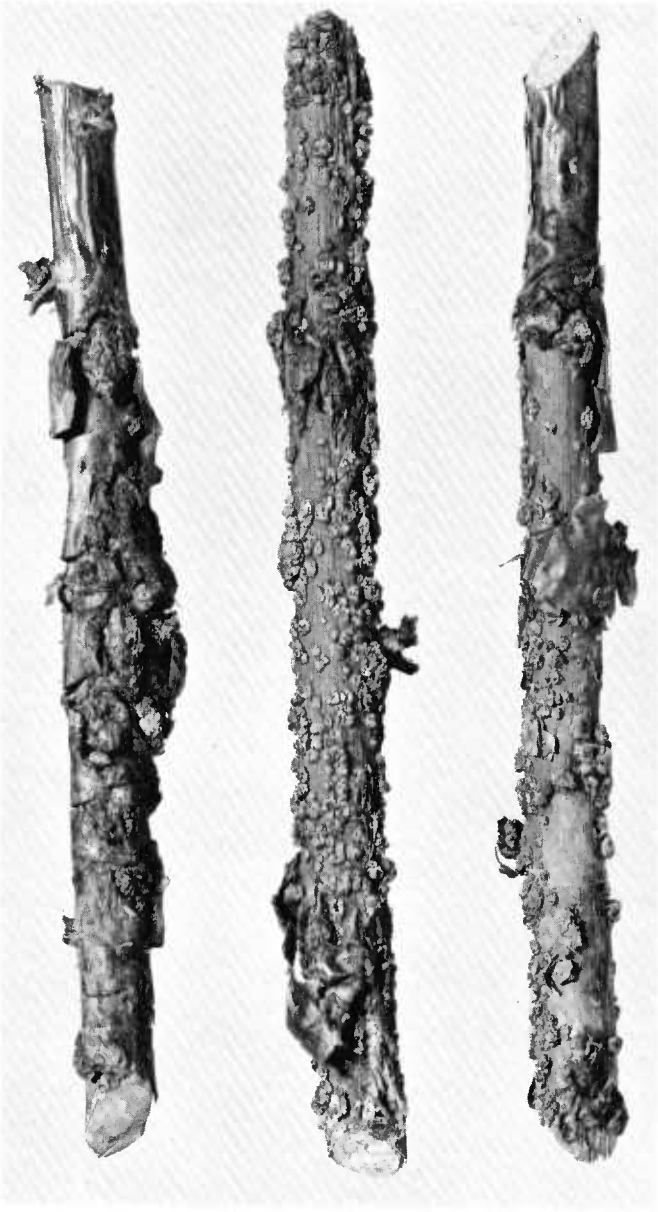

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FIGURE 26.—Cane gall of purple raspberry.

Crown Gall [24]

Crown gall is a widespread and serious disease wherever brambles are grown. The disease is one of the limiting factors in the nursery production of red raspberries (*25*, *140*). The bacteria causing crown gall have a wide host range but cause economic losses particularly on brambles, roses, and stone and pome fruits. There is a voluminous literature on crown gall because of its value as a research tool in the study of plant galls or tumors as well as its direct damage to crop plants. The reviews by Hildebrandt (*163*) and Riker et al. (*251*) and the papers in the "Symposium on Plant Tumors" (*249*) should be consulted for basic studies of the disease. Banfield (*13*) studied in detail crown gall of red raspberry.

Symptoms.—Rough, spongy, or hard wartlike growths on roots and crowns of raspberries and blackberries at the site of wounds are usually caused by crown gall bacteria, but natural overgrowths due to wounding also occur. Galls vary from the size of a pinhead to several inches in diameter. In red raspberry, galls are found mostly on the roots (fig. 27). Galls on the crowns predominate in black raspberry; abundant galls on black raspberries can reduce the production of new canes. In purple raspberry, both roots and crowns may be invaded but more commonly the latter. The tops of bramble plants often show no symptoms; in more severe cases tops may make stunted growth, in which case the plants should always be dug up for positive identification.

Causal organism.—The bacteria causing crown gall are Gram-negative rods, 0.7μ to 0.8μ x 2.5μ to 3.0μ, occurring singly or in pairs, encapsulated, and motile by means of 1 to 4 flagella. On agar, *Agrobacterium tumefaciens* colonies are small, circular, and white, and the bacteria are able to form nitrite from nitrate to a slight extent, a primary characteristic distinguishing this species from *A. rubi* (Hildebrand) Starr & Weiss (the cane gall organism, p. 54), and from *A. rhizogenes* (Riker et al.) Conn (the hairy root organism), which has been reported on red raspberry (*305*) but is primarily a pathogen of apple. Unlike *A. rubi*, *A. tumefaciens* can utilize asparagine as a sole source of carbon (*40*). Absorption of aniline blue and growth on sodium selenite agar also distinguish *A. tumefaciens* from *A. rhizogenes* (*40*).

Some strains of *Agrobacterium tumefaciens* produce a bacteriocin (agrobacteriocin 1) that is an effective antibiotic against some other strains of *A. tumefaciens* and some other bacteria (*286*). Filterable L forms of *A. tumefaciens* probably occur (*203*).

Identification of *Agrobacterium tumefaciens* is usually accomplished by isolation on Patel's medium (*234*) followed by pathogenicity tests of presumptive *A. tumefaciens* colonies on suitable plants, as tomato (*102*). Members of the genus *Agrobacterium* are said to be unique in producing 3-keto glycosides. An agar-plate technique has been devised for the production and detection of 3-keto lactosides by formation of a yellow ring of cupric oxide around *Agrobacterium* colonies grown on lactose agar flooded with Benedict's solution (*30*).

[24] Caused by *Agrobacterium tumefaciens* (E. F. Smith & Town.) Conn.

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FIGURE 27.—Crown-gall-infected red raspberry. (After Fulton, 1960.)

Presumably this technique would not distinguish among the different *Agrobacterium* species, but there is no direct evidence of 3-keto glycoside production by *A. rubi*.

Races of *Agrobacterium tumefaciens* are reported from Britain, one of them on brambles (*340*). Most isolates of *A. tumefaciens* have a wide host range.

Bacteriophages able to lyse *Agrobacterium tumefaciens* cells are reported (*44*, *88*, *222*, *232*); but at least some of these phages are able to lyse bacteria other than *A. tumefaciens* (*88*, *232*), and clear-cut techniques for identifying or controlling this bacterium by means of specific phages have not been developed.

Disease cycle.—Crown gall bacteria are wound parasites, able to gain entry into the host only through ruptures in the epidermis or bark of roots or crowns (*13*). The bacteria can invade roots and crowns from infested soil through natural growth cracks, through tissues damaged by winter weather, or through tissues damaged by soil insects (*25*). Much infection also develops during pruning and cultivation (*73*). Healthy nursery stock held in infested storage areas or mixed with diseased stock can become infected (*13*, *14*). After invasion, an incubation period of 11 to 28 days, more if the stock is dormant, may be required before the bacteria induce cell proliferation, enlargement, and disorganized growth, resulting in the production of galls (*13*). The physiology of gall formation is still an area of active investigation after many years (*19*, *196*, *197*, *249*).

Bacteria are abundant in outer portions of galls, which are continuously being sloughed off into the soil (*13*), where they may persist for 5 months (*235*) or longer, depending on soil conditions (*102*). The absolute length of time *Agrobacterium tumefaciens* can persist in soil in the field is not known, but there is as yet no good evidence that the organism can multiply in the soil (*102*). Factors that decrease the population of *A. tumefaciens* in the soil are warm temperatures, dry soils, and soils with low pH values (*102*). Sandy soils increase crown gall development (*25*, *28*), perhaps because of fewer competitive and antagonistic micro-organisms present in sandy as compared with clay soils. When such competition is not a factor, *A. tumefaciens* populations decrease more rapidly in coarse- than in fine-textured soils (*102*). Crown gall bacteria are more abundant in the top 4 inches of soil than at lower levels (*102*). In the raspberry, bacteria can persist in galls from year to year (*250*).

Host resistance.—Standard commercial raspberry varieties are almost uniformly susceptible to crown gall. However, the minor red raspberry varieties Surprise and Van Fleet are reported to be somewhat resistant (*90*, *92*).

Control.—Healthy plants in uninfested soils do not develop crown gall. Clean stock and clean land are therefore the two keys to the practical control of the disease. Only planting stock certified to be free from crown gall by a State nursery inspection service should be grown. Stock should be examined before planting to check for the development of incipient galls that might have been latent or too small to be seen during the nursery inspection. Fields that have recently been in vegetables or grain are good planting sites for brambles; fields previously in brambles, grapes, or tree fruits should be avoided, as also should infested fields that harbor large numbers of root-attacking soil insects. Fields known to be infested with the crown gall bacterium should be planted to nonsusceptible crops for 2 or 3 years or more before being planted to brambles (*25*, *28*). More data are needed from infested fields with various histories, particularly with regard to the survival of bacteria in galls in the soil.

Unnecessary root wounding by too close cultivation favors crown gall development and should be avoided. Root-feeding insects, and possibly other organisms that cause root wounds through which crown gall bacteria might enter and infect bramble plants, should be controlled. Banfield (*13*) considers that injuries from soil arthropods are the major sites of crown gall invasion. Granovsky (*141*) reports a good example of the development of severe crown gall in raspberries planted in Minnesota after a sod crop that harbored large numbers of root-attacking insects. Some raspberry root-feeding insects are rather easily controlled (*318*).

A few crown-gall-infected plants in an otherwise healthy field can be successfully dug and burned. A soil drench of formaldehyde has been recommended in West Virginia for disinfesting small areas (*245*). Methyl bromide fumigation under tarpaulin has also been found to be effective (*103*, *223*), but only if galls are first allowed to decompose in the soil. Some commercial fumigation for crown gall control is already practiced by some tree-fruit growers, as in New York (*103*), but not yet on brambles in that State (*266*).

Crown gall can be readily spread by pruning shears when pruning in the field and when trimming dormant plants for storage. Frequent dipping of pruning tools in formaldehyde has been suggested to reduce spread of crown gall bacteria (*245*).

The system of controlling crown gall infection in dormant tree-fruit stocks by soaking in disinfectants, such as organic mercurials (*98*, *158*) and antibiotics (*7*, *36*, *97*, *98*), has been proposed for bramble stocks (*158*); but such practices have not actually been investigated with raspberries. The background is prepared for valuable research on dip and drench treatments for control of crown gall in the raspberry nursery operation.

Heavy applications of nitrogenous fertilizer promote growth and improve yield in raspberry fields where crown gall is too widespread for practical roguing to be done. Heavy fertilization sometimes enables a grower to lengthen the productive life of a badly infested raspberry field for a few years (*266*).

Fire Blight [25]

Fire blight has been reported on Latham red raspberry in Maine (*125*, *126*, *282*) and on raspberry and blackberry in North Carolina (*206*). The disease was first reported on *Rubus* by Lehman (*206*). It is characterized by death of stem tips and of leaves and blossoms (*206*). Fire blight on raspberries is insect spread and rare (*126*), and is worse in rainy weather (*126*). It will not cross-infect apple (*40*, *206*, *282*).

[25] Caused by *Erwinia amylovora* (Burr.) Winslow et al. f. sp. *rubi* Starr et al.

DISEASES OF ERECT AND TRAILING BLACKBERRIES

Virus Diseases

Many of the virus diseases affecting raspberries have been found in blackberries. Often, infected blackberries are symptomless. The effects of raspberry viruses on blackberries are either unknown or the viruses occur so infrequenctly that they are overlooked. Until more is known of the attack on blackberries by the viruses that attack raspberries, growers would do well not to plant blackberries near raspberries. The literature on virus diseases of blackberries is reviewed by Stace-Smith (*276*) and Smith (*269*). This group of blackberry diseases has received comparatively little attention.

Five blackberry virus diseases known in the United States on which some studies have been made will be considered; a miscellaneous group, mostly studied abroad, is briefly discussed.

Blackberry Mosaic

Alcorn et al. (*4*) described a disease of Himalaya blackberry (*Rubus procerus* P. J. Muell.) in California having chlorotic spots, feather-vein chlorosis (fig. 28), and oakleaf patterns on leaves, the plants being dwarfed and finally dying. The virus causing this blackberry mosaic, or the putative component viruses causing it, may possibly occur in Himalaya blackberry in symptomless condition (*4*). The disease is presumably quite distinct from the aphidborne raspberry mosaics, which have been found in blackberries (*24, 56, 81*).

The experimental host range includes several other erect and trailing blackberries, Lloyd George raspberry, almond, and peach; bud failure was induced in peach (*4*). Blackberry mosaic was soil borne to peach in a low percentage of cases. Alcorn et al. (*4*) felt on the basis of host range and cross-inoculations that blackberry mosaic involves peach yellow bud mosaic virus and probably an additional virus component.

Peach yellow bud mosaic virus was later shown to be soil borne to peach by the nematode vector *Xiphinema americanum* Cobb (*39*), and to be closely related serologically and by cross-inoculations to tomato ringspot virus (*61*).

Since the work of Alcorn et al. (*4*), blackberry mosaic virus has been transmitted mechanically to peach seedlings (*193, 342*), where it interferred with the movement of the typical peach yellow bud mosaic virus that had been previously introduced. A large number of herbaceous hosts were successfully inoculated with blackberry mosaic virus, by mechanical transmission from Himalaya blackberry and red raspberry. This commercial raspberry stock, on which vein-feathering mosaic and ringspots were seen, was found in a California field previously planted to peaches infected with yellow bud mosaic virus. These isolates are regarded as being strains of peach yellow bud mosaic virus (*193*).

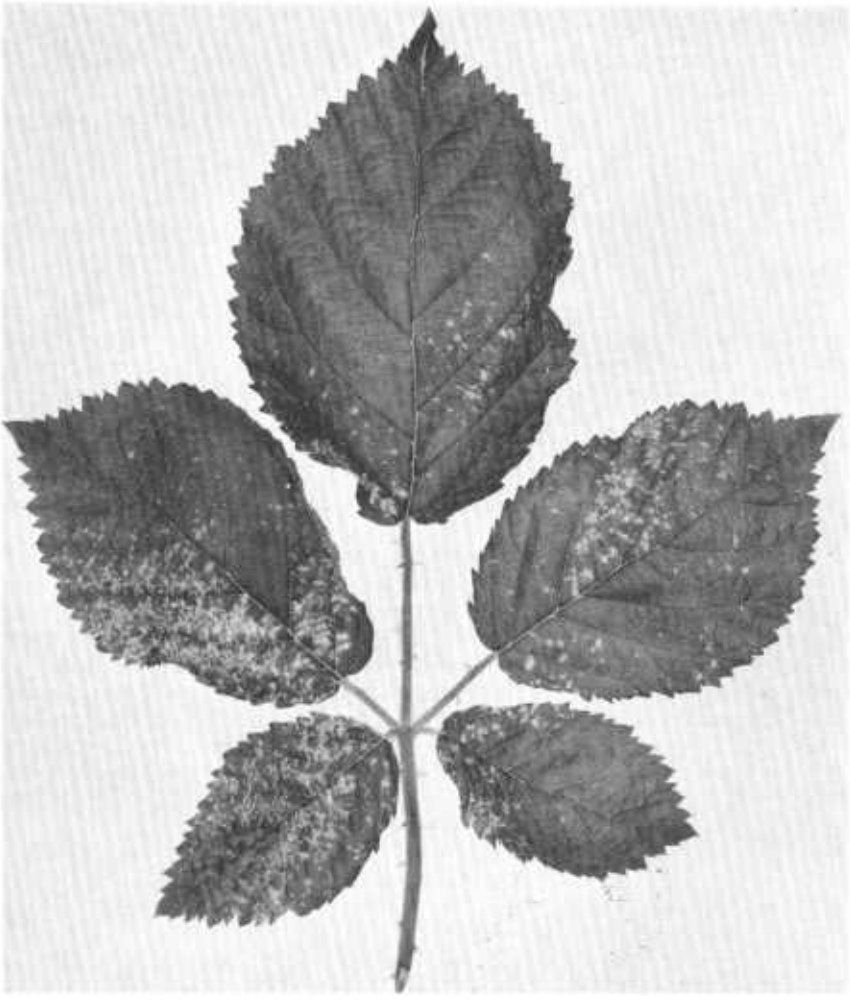

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FIGURE 28.—Blackberry mosaic in Himalaya blackberry leaf.
(Courtesy of University of California, Berkeley.)

The vector relationships of blackberry mosaic remain to be determined directly. The possibility of more than one virus entity being present (*4*) complicates these relationships. Not determined are the serological relationships of these viruses to tomato ringspot virus, or to the raspberry ringspot virus (with a nematode vector [26]) that Stace-Smith considers to be a strain of tomato ringspot virus (*280*). It seems quite possible at this juncture that a strain or strains of tomato ringspot virus are involved in the California blackberry mosaic as well as the British Columbia raspberry ringspot.

[26] Stace-Smith, R. Personal communication, 1964.

Calico

Calico, a virus disease of Loganberry, exhibits yellow blotches of the leaves. Infected plants are not severely damaged if they are grown under irrigation (*332*) (fig. 29). The natural method of spread of calico is unknown, but it is widespread in old Loganberry fields in California (*332*). Use of stock free from calico virus, roguing out infected plants, and elimination of wild *Rubus ursinus* Cham. & Schlecht. near plantings are recommended control measures.

Dwarf

Zeller (*348*) described a virus diesease of Loganberry and Phenomenal that was widespread along the Pacific coast, occasionally causing severe damage to individual plantings; he called it blackberry dwarf. Infected plants had dwarfed light-green, mottled, obovate leaves and canes with short internodes. *Chaetosiphon* (*Capitophorus*) *tetrahodus* (Wlk.) from roses was found to be a vector under experimental conditions (*348*). Boysenberry and the blackberry variety Young (Youngberry) were later found to be essentially symptomless hosts (*358*).

Wilhem et al. (*331*) described Loganberry dwarf as a new virus disease that is important in California Loganberry production (*332*). Infected plants have weak, spindly canes that bear witches'-brooms of leaves that cup downward and become reddened or bronzed prematurely in the fall (fig. 30). Severely infected plants do not tip-root. Within 3 years infected plants become completely unproductive. Loganberry dwarf has been recovered from symptomless *Rubus*

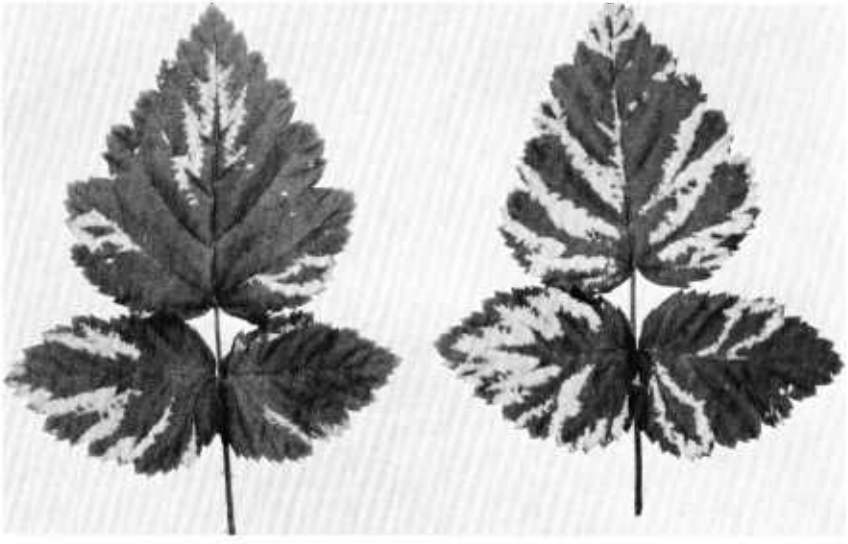

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FIGURE 29.—Calico disease of Loganberry. (After Wilhelm et al., 1951.)

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FIGURE 30.—Dwarf disease of Loganberry; on right, healthy cane. (After Wilhelm et al., 1951.)

ursinus Cham. & Schlecht., the wild Pacific coast blackberry (California trailing blackberry) (*331*). Experimentally susceptible to Loganberry dwarf, some varieties remaining symptomless, are the blackberries Boysenberry, Cory's Thornless, Nectarberry, Phenomenal, and Youngberry, and some raspberries (*331*). The natural method of spread of Loganberry dwarf is unknown.

The two diseases, blackberry dwarf described by Zeller (*348*) and Loganberry dwarf described by Wilhelm et al. (*331*), have some hosts in common, although there are differences in described symptoms. Additional studies will be required to establish their relationship.

Recommended control measures for the blackberry and Loganberry dwarf diseases are use of clean stocks and the prompt removal of infected plants, also destruction of sources of infection in nearby areas. Phenomenal is reported as a reservoir of blackberry dwarf (*348*) and *Rubus ursinus* of Loganberry dwarf (*331*).

Sterility

Plants of many blackberry varieties produce excellent foliage and normal-appearing flowers that fail in whole or in part to set fruit (fig. 31). Hereditary abnormalities and insect damage account for some of this difficulty (*266*), but graft-transmissible sterility has also been shown to occur in erect and trailing blackberries and black rasp-

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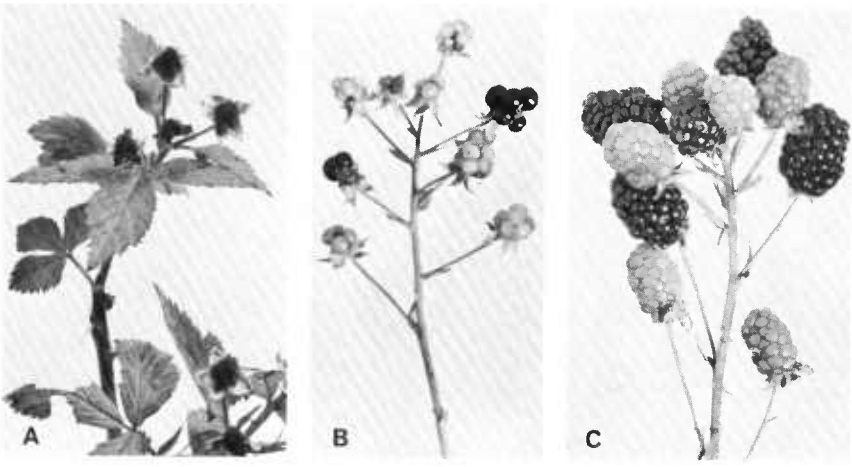

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FIGURE 31.—Sterility disorder of blackberry: *A*, Completely sterile fruits; *B*, partially sterile fruits; *C*, normal fruits.

berries (*160*). The transmission of sterility by grafting indicates the viral nature of the disorder. The virus spreads in the field, but its means of spread, other than by root suckers (*160*), has not been determined. Sprays of dieldrin and DDT at bloom did not prevent development of the disease (*160*). Pollen from sterile plants germinated normally (*220*).

Infected blackberries usually grow more vigorously than healthy plants (*220*) and are, therefore, often chosen for propagation. The Alfred, Eldorado, and Lawton varieties are particularly notable for being contaminated with the sterility virus. Viral sterility probably occurs in many parts of the United States in *Rubus* (*159*, *160*, *220*, *221*).

Treatments in hot water at 50° C. and in hot air at 38° to 42° did not eliminate the virus from infected plants (*160*).

In order to control virus-induced blackberry sterility, all plants that fail to set fruit should be removed by grubbing out the plant and as much of the root system as possible. Root suckers for propagation should be dug only from fields where the sterility condition has not been previously found. Plants should be purchased only from those nurseries that will certify that their plants were propagated from fruitful stock.

Variegation

A variegation on one Maryland farm was reported by Horn in 1948 (*167*) from wild *Rubus allegheniensis* Porter. The symptoms ranged from white islands of leaf tissue to nearly white leaves when grafted to blackberry or black raspberry. Other information on this virus is

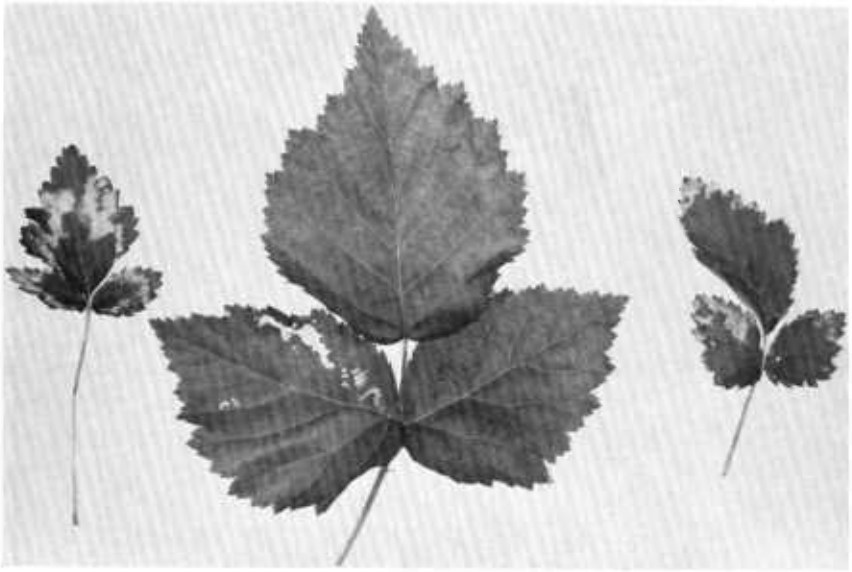

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FIGURE 32.—Variegation of blackberry.

lacking, but a clump of *R. allegheniensis* with a similar variegation (fig. 32) was found in the same locale in 1960 by the author.

Other Blackberry Virus Diseases and Viruslike Disorders

In Stanislaus County, Calif., in 1956 the Olallie blackberry variety was reported to be showing symptoms of leaf yellowing, dwarfing, and shoot proliferation (*230*). No transmission or other studies of this disorder were reported.

Cadman (*56*) noted that several viruses occurred in blackberry varieties in Scotland; these produced severe stunting and necrosis when the varieties were grafted onto the red raspberry variety Norfolk Giant. The viruses could then be moved to black raspberry seedlings by means of the aphid *Amphorophora rubi*. Viruses latent in raspberry were found to cause severe necrosis and stunting in blackberry. One or more viruses causing vein clearing and oak leaf patterns in black raspberry seedlings, but not transmissible by *A. rubi*, were found by the author in Lawton variety of blackberry in Maryland.

Rubus stunt virus, transmitted in Europe by the leafhopper *Macropsis fuscula* Zett., occurs in certain wild European blackberries, which can serve as virus reservoirs and as vectors for cultivated raspberries (*124*). Rubus stunt is not known in the United States. (See p. 21.)

Legg (*205*) described a virus disease, caused by the Loganberry degeneration virus, that is detectable only by grafting to *Rubus henryi* from weakened Loganberry plants. Infected plants are symptomless, but yield and cane production were reduced 36 and 34 percent, respectively, below comparable values from vigorous virus-tested stock.

Fungus Diseases

Anthracnose [27]

The same fungus that causes anthracnose of raspberry plants (see p. 21) also attacks erect blackberries and trailing blackberries (dewberries). In the Southeast, anthracnose is severe in erect and particularly in trailing blackberries. In this region, anthracnose control is one of the major factors in successful blackberry production.

Symptoms.—A few days after the fungus begins to invade the living bark of young blackberry canes, minute purplish spots appear. These spots increase in size, turning light gray at the center, and the margin becomes purple brown. The lesions may run together and become elongated and irregular in outline. (fig. 33, *B*). The diseased tissue

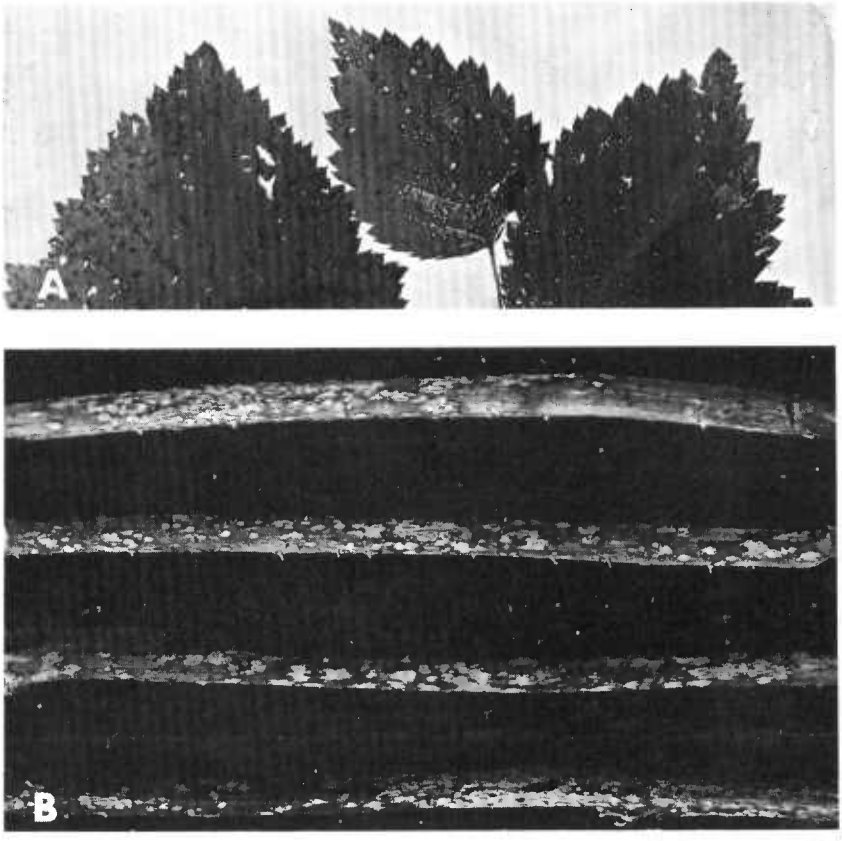

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FIGURE 33.—Anthracnose of Lucretia trailing blackberry: *A*, Leaves; *B*, canes.

[27] Caused by *Elsinoë veneta* (Burkh.) Jenkins; imperfect stage is *Sphaceloma necator* (Ell. & Ev.) Jenkins & Shear.

extends down into the bark, partly girdling the cane and interrupting the flow of sap. As the cane dries out, it may crack.

In leaves the spots are at first very small, but by their running together large grayish dead areas are formed between the veins (fig. 33, *A*). On leaves of trailing blackberry infected areas may drop out to produce shotholing. Anthracnose does not usually cause much damage to leaves of erect blackberry. Large leaf spots with white centers containing minute black pycnidia are caused by *Septoria rubi* West. (p. 78), not by the anthracnose fungus.

The berries of most varieties of erect blackberries are fairly resistant to anthracnose infections. On trailing blackberries immature drupelets are often badly infected. Scabby anthracnose spots on such drupelets increase in size and prevent normal ripening, causing the fruit to be small, brown, dry, and woody (see fig. 36). Drupelets attacked when more mature are brown and sunken (*338*). Much loss to trailing blackberries in the southeastern part of the United States is due to the direct infection of the drupelets by anthracnose fungus. Scabby berries lower the value of the entire fruit pack and are very susceptible to secondary infection by other fruit-rotting fungi.

Causal organism and disease cycle.—See pages 21 to 24 for a full discussion of *Elsinoë veneta* and its life cycle on raspberries. On erect and trailing blackberries the fungus is more confined to the canes than it is on raspberries. *E. veneta* lives over the winter on infected canes. Fruit infections on trailing blackberries are favored by excessive rainfall during the time of fruit development (*338*).

Host resistance.—There are differences in varietal susceptibility to anthracnose among both the erect and the trailing blackberries. Himalaya, Lawton, and Synder erect blackberries are listed as susceptible, Evergreen (*Rubus laciniatus* Willd.) as resistant (*111*). Loganberry, Lucretia, and Mayes (Austin Mayes) trailing blackberries are susceptible but Young (Youngberry) is somewhat resistant (*95*, *111*). In areas where anthracnose is severe in trailing blackberries, no varities are available that do not require spraying and pruning.

Control.—In general, the measures used to control anthracnose on raspberry (see p. 25) are applicable to anthracnose control on erect and trailing blackberries.

Spraying for anthracnose is not always necessary where the disease is not severe. In Oklahoma on the anthracnose-susceptible blackberry variety Lawton a three-spray schedule significantly reduced the number of cane lesions but did not influence yield or fruit size (*288*). The spray program for trailing blackberries in North Carolina is lime-sulfur at the delayed dormant stage, plus two ferbam sprays before harvest and one ferbam spray afterward (*136*). In order to reduce inoculum potential, both the old and new canes of trailing blackberry are cut off at or slightly below the ground level after harvest. Canes that develop in late summer are then heavily fertilized to produce fruit the next year (*95*, *136*). In this way much of the inoculum to infect late-summer growth is eliminated. Southern trailing-blackberry growers consider removal of all aboveground growth in midsummer to be their most important cultural operation (*95*).

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Cane Blight [28]

Cane blight is a problem on Evergreen blackberry (*Rubus laciniatus* Willd.) in the Pacific Northwest (*6*). Other erect and trailing blackberries are sometimes infected. See page 26 for a fuller discussion of the cane blight fungus. Pruning below ground level is recommended for control of cane blight on Lucretia trailing blackberry in the South (*95*).

Ascospora ruborum Zeller also commonly causes cane lesions on several types of brambles in Oregon. This disease, known as cane spot, occurs on raspberry, Loganberry, and Himalaya and Evergreen blackberry. Large brown patches form on the canes in early winter, later turning gray (*347*).

Cane and Leaf Rust [29]

On certain susceptible blackberries, cane and leaf rust, also called blackberry rust and yellow rust, is of economic importance in some years in the Southeastern States and the Pacific Northwest. Cane and leaf rust is not a systemic rust but is often mistaken for the systemic orange rust caused by *Gymnoconia peckiana* (p. 72). The cane and leaf rust disease was studied in Washington State by Fischer and Johnson in 1950 (*122*).

Symptoms.—In Washington State cane and leaf rust is first seen in late spring on infected blackberry fruiting canes, which develop large lemon-yellow uredia that split open the bark (fig. 34, *B*). Small yellow uredia develop during early summer on under surfaces of leaves on fruiting canes (fig. 34, *A*); in years of severe infection this can cause premature defoliation. Uredia may also occur on the fruit, but this is rather rare. Buff-colored telial sori develop among the uredia on leaves in early fall. Pycnia and aecia also develop on lower leaves of current-season canes in October and November (*122*).

Causal organisms.—Pycnia are epiphyllous on reddish spots, large, prominent, and pustular. Orange-yellow aecia surround the pycnia, often in confluent rings. Aeciospores are globoid or obovoid, 18μ to 19μ x 19μ to 23μ, the walls colorless, closely verrucose, 2μ to 2.5μ thick, with obscure pores. Uredia are hypophyllous, scattered, powdery, lemon yellow colored when fresh. Old parasitized uredia may be white. Urediospores are obovoid, 16μ to 19μ x 21μ to 27μ, the walls nearly colorless, finely and closely verrucose-echinulate, 1.5μ to 2μ thick, with 3 to 4 indistinct equatorial pores. Telia are hypophyllous, scattered among uredia on old leaves, pale buff. Teliospores are cylindrical, 18μ to 24μ x 85μ to 110μ, 5- to 13-celled, irregularly flattened or coronate above, narrow below, each cell extending into a pore-bearing tip next to the cell above; walls colorless, 1.5μ to 2μ thick on the sides, thicker above, smooth or slightly roughened at the apex. Pedicels are colorless and very short to apparently lacking (*9*, *122*).

Disease cycle.—Urediospores from fruiting-cane lesions infect leaves on fruiting canes during the growing season. Severe infection from

[28] Caused by *Melanomma coniothyrium* (Fckl.) L. Holm (=*Leptosphaeria coniothyrium* (Fckl.) Sacc.); imperfect stage is *Coniothyrium fuckelii* Sacc.
[29] Caused by *Kuehneola uredinis* (Lk.) Arth.

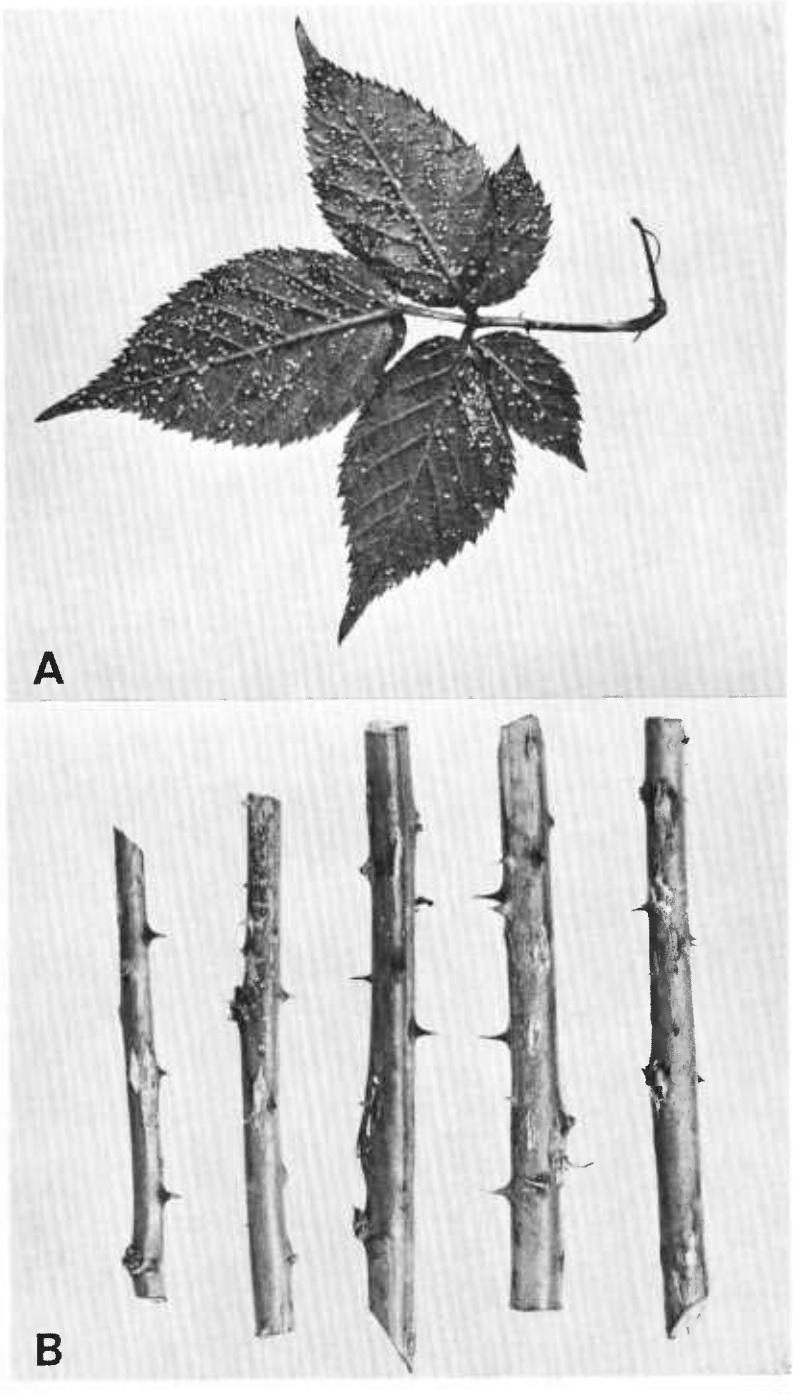

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FIGURE 34.—Cane and leaf rust of blackberry: *A*, Leaf; *B*, canes.

leaf to leaf is favored by wet summer weather. Telia develop on fruiting-cane leaves in the fall, and sporidia from germinating teliospores infect adjacent leaves of current-season canes, where pycnia and aecia are formed. The fungus probably overwinters on canes as uredial mycelium or as latent uredia, but the details of cane infection and overwintering have not been studied (*122*). Not investigated is the degree to which *Kuehneola uredinis* can damage the canes, nor the possible role of the cane blight fungus *Melanomma coniothyrium* in intensifying damage, as happens in yellow rust of raspberry, caused by *Phragmidium rubi-idaei* (*349*, *351*). (See p. 53.)

Host resistance.—*Kuehneola uredinis* is known only on *Rubus* species, mostly of the *Eubatus* section. The following wild species are susceptible according to Arthur (*9*) : *R. argutus* Lk., *R. canadensis* L., *R. cuneifolius* Pursh, *R. floridus* Tratt., *R. frondosus* Bigel., *R. hispidus* L., *R. lucidus* Rydb., *R. nigricans* Rydb.. *R. nigrobaccus* Bailey, *R. pergatus* Blanch., *R. plicatifolius* Blanch., *R. procumbens* Muhl., *R. pubescens* Raf., *R. recurvans* Blanch., *R. rubisetus* Rydb., *R. trivialis* Michx., *R. ursinus* Cham. & Schlecht., and *R. ursinus* var. *vitifolius* (Cham. & Schlecht.) Focke. Both red and black raspberry are recorded hosts (*305*) but are not commonly found infected. *Rubus leucodermis* Dougl., the western wild black raspberry, is not infected (*122*).

The erect blackberry variety Evergreen (*Rubus laciniatus* Willd.) was found to be susceptible in Washington State, as also were *R. macropetalus* Dougl. (the Pacific coast trailing blackberry) and the trailing blackberry variety Chehalem (*122*). Blackberries found resistant there included some trailing varieties and the erect variety Eldorado (*122*). In Maryland, Darrow (*93*) found Lawton and Eldorado blackberries to be very susceptible. Generally found resistant in Maryland were European blackberries, the Pacific coast trailing blackberry, and Evergreen (*93*). It seems probable that the differences reported in susceptibility in the same varieties in Maryland and in Washington State may be accounted for by physiologic specialization in *Kuehneola uredinis*.

Control.—Pruning and removal of old diseased canes after fruiting are desirable sanitation practices. In Oregon, three applications of spray are recommended for cane and leaf rust control (*118*) : Lime-sulfur in winter, and fixed copper at the green-tip stage and again just before blooming. Spraying and sanitation have not provided satisfactory control in Washington (*144*).

Care must be taken to distinguish between blackberry plants infected with the systemic orange rust fungus, *Gymnoconia peckiana* (see p. 72), which has pustules of orange waxy aeciospores, on leaves only, and those infected with cane and leaf rust fungus, *Kuehneola uredinis*, which has yellow pustules of powdery urediospores on fruiting canes as well as on leaves.

Fruit Rots

Erect and trailing blackberries, especially if poorly handled, are susceptible to the fruit rots described for raspberries (p. 28) (fig. 35, *A*, *B*, *D*). The quality of blackberries shipped to market can be im-

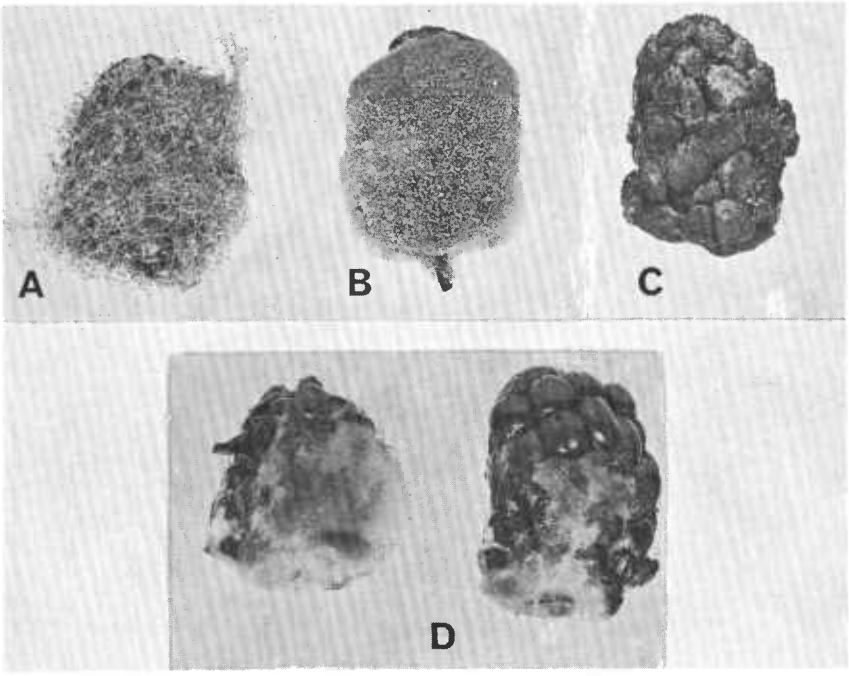

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FIGURE 35.—Fruit rots of Lucretia trailing blackberry: *A*, Rhizopus rot; *B*, gray mold; *C*, black rot; *D*, Alternaria rot.

proved by using the same techniques of handling and harvesting described for raspberries (p. 31). Certain fruit rots and fruit disorders of blackberries deserve special mention.

Botrytis cinerea is an important fruit rot of blackberries. In Oregon, it can cause a bud blight as well as a fruit rot (*305*).

Trailing blackberry fruit from the Southeast, particularly subject to anthracnose (p. 66), should be culled to remove the brown scabby (anthracnose-infected) berries from the pack before shipment (fig. 36); this will help to prevent the establishment of the common storage-rot fungi on these berries. McKeen (*213*) reviewed the literature on the dry rots of Loganberry fruit in Canada and described a new species, *Rhizoctonia rubi* McKeen, causing fruit rot of Loganberry in British Columbia. He believes that *R. rubi* rather than *Haplosphaeria deformans* (p. 81) is responsible for the dry berry condition in Loganberry.

If overripe trailing blackberries are included in the pack on the Atlantic coast, they are susceptible to black rot in transit (fig. 35, *C*). Black rot is caused by *Phyllostictina carpogena* Shear (*264*) and is also recorded on red raspberry (*305*). Black rot develops slowly and is not likely to appear on fruit until after it has left the field (*106*).

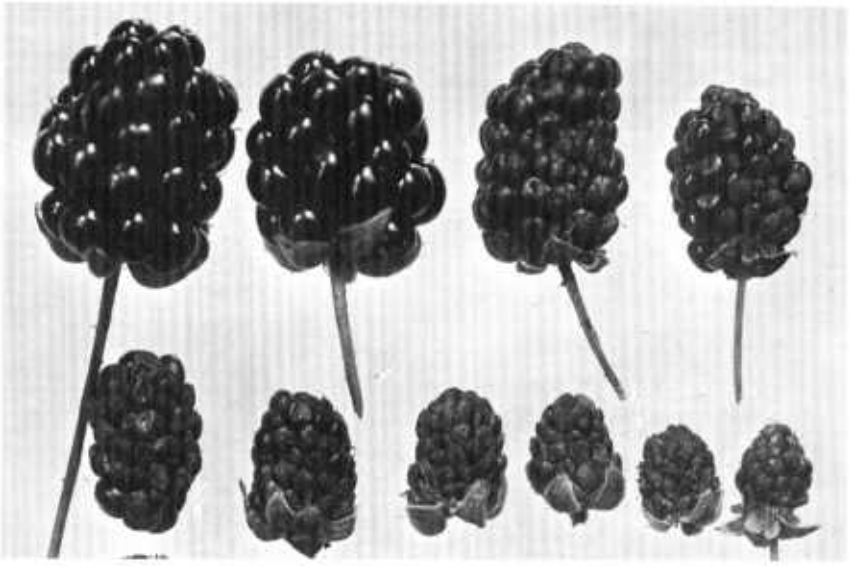

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FIGURE 36.—Anthracnose of Lucretia trailing blackberry fruits as compared with two berries, in upper left-hand corner, not diseased. (After Wolf and Dodge, 1926.)

In redberry disease, some fruits are still red when others have matured. Also, some individual drupelets of mature fruit may remain white, later rotting (*111*). Redberry is caused by the blackberry mite *Aceria* (=*Eriophyes*) *essigi* (Hassan) on the Pacific coast (*114*, *117*). The mite causes damage on Himalaya and Evergreen blackberries and is controlled by a fall spray of summer oil emulsion or a delayed spray of lime-sulfur, or by both in severe cases (*118*).

Orange Rust [30]

Orange rust is a very common and serious disease of erect and trailing blackberries throughout the United States.

Symptoms.—Orange rust appears in the spring on leaflets of systemically infected shoots after they unfold; the rust continues to develop, on leaves and especially on new canes, for 4 or 5 weeks. The young shoots are rather spindly and clustered, and their leaves are yellowish green, becoming covered with minute reddish spermogonia. In 2 or 3 weeks the undersides of the leaves will be covered with blisterlike orange-colored aecial pustules filled with waxy aeciospores (fig. 37, *A*, *B*). When dry, the spores may fall out and cover the leaves below. Later, the tips of the infected young canes seem to grow away from the fungus and recover, so that late in July or August, even in the Northern States, no orange rust will be found in the field (*108*). The infected canes, however, never reach the size

[30] Caused by *Gymnoconia peckiana* (Howe) Trott.

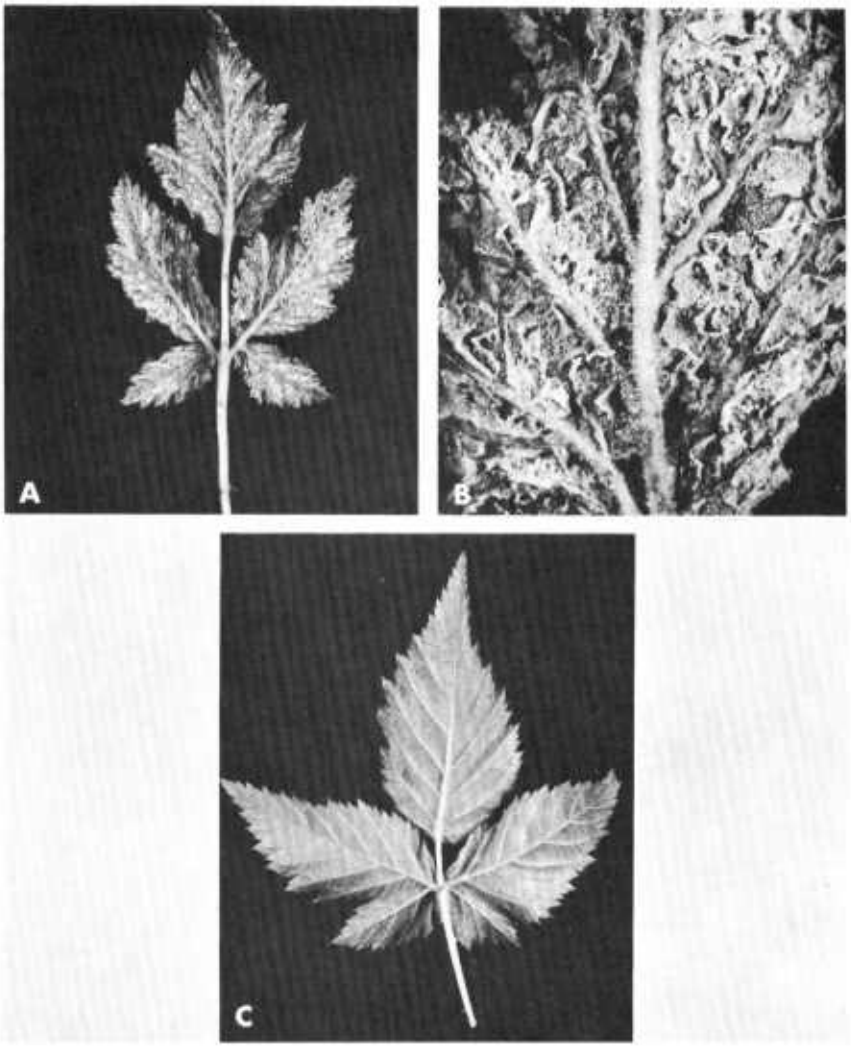

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FIGURE 37.—Orange rust of blackberry: *A*, Infected leaf showing aecial pustules; *B*, enlarged portion of infected leaflet; *C*, healthy leaf.

of normal canes. In the following years infected canes are bushy, spindly, and bear no fruit (fig. 38).

Causal organism.—The usual *Gymnoconia* form of orange rust that attacks blackberries is the form also found on black raspberries. (See p. 36 for a fuller discussion.) There is in addition a microcyclic form of *G. peckiana*, known as *Kunkelia nitens* (Schw.) Arth., lacking teliospores; in this form aeciospores function as teliospores. Aeciospores of the *K. nitens* form of the fungus on blackberries are morphologically distinct from those of *G. peckiana* (*202*), but intergrades

BN-26732

FIGURE 38.—Orange rust of blackberry, showing witches'-brooming.

and cross infection exist (*108*, *109*). *Kunkelia nitens* aeciospores, functioning as teliospores, germinate to produce 4-celled promycelia that form sporidia.

Aeciospores collected from blackberry are often of the microcyclic form but are able to infect black raspberry (*8*, *108*).

Disease cycle.—Orange rust spores are shed from blackberry leaves in the spring; shoots breaking through the ground are susceptible at this time. The plants usually become systemically infected through local infections developing from the orange rust spores on the young shoots. The fungus also grows down into the crown and out into the roots, as another means of systemic infection of the new shoots that spring up each year.

In trailing blackberries, all of which tip-root, the orange rust mycelium follows the tips as they root and then develops in the new root systems (*108*).

Occasionally a new shoot from an old crown is attacked by the fungus. The canes from the rest of the hill will continue to remain uninfected and will bear fruit. The rust may thus live several years in such a plant before all the canes show rust. A new shoot from an infected root may grow up the first spring into what looks like a normal cane, which will blossom the next year with only the leaves at the base showing rust. The following year, however, most of the canes in this hill will be found to be dwarfed and bushy, with spindly canes and without blossoms (*108*).

Host resistance.—Probably none of the cultivated erect blackberry varieties is actually immune from orange rust. Youngberry, Loganberry, and the wild Pacific coast blackberry (*Rubus ursinus* Cham. & Schlecht.) are susceptible in California (*11*). The Snyder, Evergreen, and Eldorado, though very resistant, can be infected (*108*). Lawton blackberry is reported as resistant (*182*). Many wild trailing blackberries are very susceptible, but Lucretia and Boysenberry trailing varieties are immune (*14*, *108*). Pathogenic races of the rust may occur (*108*).

Control.—Rust-free nursery stock should be planted. The area around the field should be clear of infected wild blackberry and wild black raspberry. If orange rust appears on a blackberry plant during the spring or summer in which it is set out, the stock was infected when planted. Propagations for new plants should not be made from roots or canes of any hill that has shown this rust.

Plants showing orange rust should be pulled up, including the roots. This should be done before the spores are shed.

If new plantings and environs are carefully inspected about a month after planting and all rusted plants rogued, the grower will have little difficulty later in keeping his fields free from orange rust (*108*). Should a rust appear on blackberry leaves in August or September, it is not orange rust, but cane and leaf rust caused by *Kuehneola uredinis* (Lk.) Arth. (p. 68); the plants should not be destroyed.

Root Rots

For a general discussion of root rots of brambles see pages 41 and 42.

Rosette [31]

Rosette, or double blossom, is a fungus disease that seriously affects many varieties of erect and trailing blackberries. The disease occurs mainly from New Jersey to Illinois and south; it is not reported from the Pacific coast. Rosette was studied by Cook (*83*) and Plakidas (*239*).

Symptoms.—Buds of erect and trailing blackberries on new canes or vines become infected early in the summer. They show no obvious signs of infection until the following spring, unless a late warm fall follows a severe infection, when a few fall witches'-brooms develop. In the spring numerous short leafy sprouts develop from infected buds, forming little witches'-brooms with pale-green foliage that later turns bronze (fig. 39). Several of these witches'-brooms may be formed on one cane. The unopened infected flower buds are somewhat larger and coarser than usual, and frequently somewhat redder. Sepals enlarge and occasionally change into leaves. The petals are much wrinkled and twisted as they unfold; they are frequently pinkish in color and occasionally become green and leaflike. The pistils are often large and long, occasionally developing abnormal shapes. Pistils and stamens become covered with the whitish spores of the fungus. Berries do not develop from infected blossoms, although noninfected parts of the same cane may bear some poor fruit.

The disease is not manifested by witches'-brooms in all varieties; however, fruit set in infected blossoms is always impaired.

BN–26948

FIGURE 39.—Rosette of the Lucretia trailing blackberry.

[31] Caused by *Cercosporella rubi* (Wint.) Plakidas.

Causal organism.—The mycelium is hyaline, septate, 1.5μ or less in diameter. Aerial hyphae 2.0μ to 2.5μ in diameter anastomose freely to form a networklike growth. Conidiophores arising as side branches singly or in fasicles are hyaline and short, averaging 3.8μ x 12.7μ, usually unbranched and lacking septa, with 1 to 7 spore scars. Conidia are hyaline, cylindrical to tapering, straight to curved, 0- to 12-septate (average 3) with or without constrictions at septa, very variable in length, 13μ to 96μ x 2.7μ to 4.7μ (average 33.8μ x 3.8μ). The mycelial stage occurs within vegetative and floral buds of hosts, and conidia are formed in opened blossoms (*239*).

Disease cycle.—Infection takes place in the spring on young buds. The mycelium penetrates between the bud scales and surrounds the embryonic bud elements. As secondary buds develop beside an infected bud, the fungus also invades them. After infection, mycelium remains limited throughout the summer. The buds are externally symptomless, except when a few may rarely be forced out in an unusually warm late fall. These buds then produce witches'-brooms. Mycelium increases in the buds over the winter, the hyphae surrounding and being enveloped by the growing primordia but not invading them. Bud proliferation is induced. In the spring, infected vegetative buds develop into witches'-brooms. Investigation of the possible production of auxin-like chemicals by *Cercosporella rubi* should be undertaken. Infected flowerbuds produce blossoms in which the fungus sporulates heavily on stamens and pistils. The spores are windborne and carried by insects to new buds, which are susceptible to infection only in the spring, excepting one variety studied (Nanticoke), found to be infected in August (*83*, *293*).

In the case of some trailing blackberry varieties, mycelium moves from infected buds through the stem and becomes established in the crown of the rooted stem tip, where it perennates and may infect buds in new shoots (*239*).

Host resistance.—*Cercosporella rubi* is limited to the genus *Rubus*, and primarily to certain wild and cultivated erect and trailing blackberries in the subgenus *Eubatus*, although there are a few reports of its occurrence in red and one report of its occurrence in black raspberry (*305*).

Among the erect blackberries, Evergreen (*Rubus laciniatus* Willd.), Lawton, McDonald, Merserau, and Nanticoke are susceptible to rosette (*94*, *239*). Brainerd is quite resistant in North Carolina (*94*) but susceptible in Louisiana (*239*), indicating the possible existence of physiologic races within *Cercosporella rubi*. Himalaya (*R. procerus* P. J. Muell.) is immune (*239*).

Among the trailing blackberries, Lucretia, Mayes (Austin Mayes), and Youngberry are susceptible (*83*, *95* *239*), but the Advance (=Rogers?) is reported as immune (*63*).

Control.—Sanitation and spraying are the general methods of control. Wild erect and trailing blackberries in the vicinity should be destroyed so far as practicable. Infected blossom clusters in the planting can be removed by handpicking before they open, where there is sufficient farm labor to do this. Trailing blackberries are often cut off at the ground after fruiting in Delaware and southward, where the growing season is long. In this way infected buds are removed.

Since spores that cause new infections are found only in flowers, subsequent new growth that year will be free from rosette (*136*, *239*). In northern regions, where sufficient new growth cannot be obtained if all vines are cut off after harvest, only the old vines should be cut out and burned. It will then be necessary to handpick and burn the infected blossoms as they appear the following spring, or to spray. Excellent control of rosette was obtained in Louisiana by spraying three times with bordeaux mixture during the flowering period (*239*). It is probable that captan would also be effective for this purpose, but data are not available on this point.

The partial to complete sterility of certain flowers or of the whole plant because of insect damage or because of a virus disease (see blackberry sterility, p. 63) should not be confused with the rosette disease, where the whitish masses of *Cercosporella rubi* conidia are readily seen in infected flowers.

Septoria Leaf Spot [32]

Leaves and canes of erect and trailing blackberries are frequently badly spotted by Septoria leaf spot, also called leaf and cane spot, in the Southeastern States and the Pacific Northwest. The life cycle of the fungus and its perfect stage are described by Roark (*252*); the perfect stage he described could not be found by Demaree and Wilcox (*100*). The disease occurs in Great Britain (*147*). *Rhabdospora ramelis* (Rob. and Desm.) Sacc. also causes a leaf spot of blackberry in Great Britain known as purple blotch (*341*).

Symptoms.—On leaves Septoria leaf spot lesions have a whitish center and a brown or purple border (fig. 40). The lesions are more circular in outline and develop later in the season than anthracnose spots, with which they are often confused. Septoria leaf spot lesions are larger, being about 3 or 4 mm. Small black pycnidia can be seen in the central portions of the lesions. Spots on canes and petioles are elongate in outline (fig. 41). Premature defoliation and winter injury are the important consequences of heavy infection.

Causal organism.—The conidial stage, *Septoria rubi* West., produces brown to black, flattened, epiphyllous pycnidia with wide ostioles. Conidia are hyaline, filiform, 40μ to 55μ x 1.5μ, obscurely 2- to 3-pluriseptate (*252*). The conclusions of Demaree and Wilcox (*100*) have been adopted in this handbook, namely that *Septoria rubi* West. is considered the main leaf-spotting fungus on erect and trailing blackberries and that *Sphaerulina rubi* Demaree & M. S. Wilcox is the main one on raspberries. (See p. 35 for synonomy of *Sphaerulina rubi* as concerning raspberry.)

A description follows of the ascigerous stage according to Roark (*252*), whose work could not subsequently be repeated (*100*): Perithecia are mainly hypophyllous, gregarious, erumpent, globose, 60μ to 80μ in transverse diameter, lacking paraphyses, with short papilliform ostioles. Perithecial walls are black, pseudoparenchymatous, 2 or 3 cells thick. Asci are subclavate to cylindrical, very short-pedicillate, 8-spored, irregularly biseriate, 45μ x 8μ to 10μ in water. Ascospores are hyaline, slenderly fusiform, straight or slightly curved,

[32] Caused by *Septoria rubi* West.

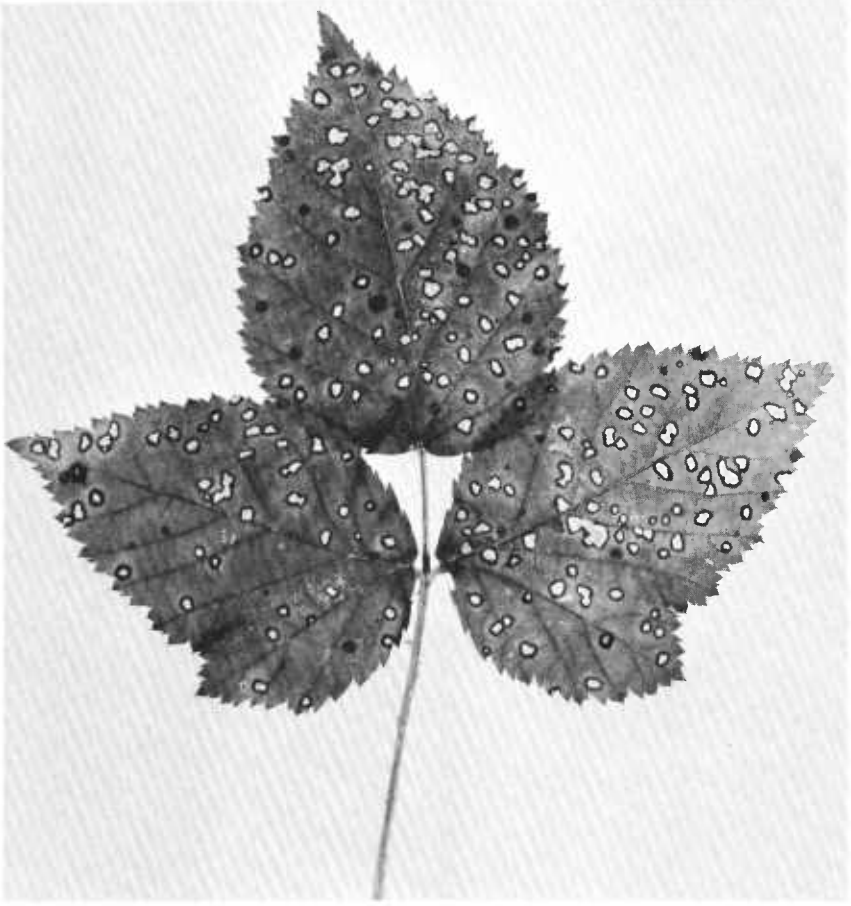

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FIGURE 40.—Septoria leaf spot of trailing blackberry.

1-septate with a very slight constriction at the septum, 20μ to 25μ x 3.5μ to 4.3μ, extreme dimensions in length being 17μ to 28μ.

In the Southeastern United States *Mycosphaerella confusa* Wolf (imperfect stage is *Cercospora rubi* Sacc.) (*337*) also occurs, on erect blackberries and particularly on trailing ones, causing a disease known as leaf spot or blotch (*305*).

Disease cycle.—According to Roark (*252*) the fungus overwinters as mycelium and immature winter pycnidia in dead leaves and stems, and to a lesser extent as perithecia. Pycnidia on canes of nursery stock are a common means of movement of the fungus into new fields. Conidia released in masses from pycnidia are moved to infection courts by splashing or wind-driven rain. Ascospores are formed from May to July in Wisconsin. Secondary infections continue throughout the growing season and are related to periods of rainfall (*252*). Although the ascigerous stage is reported to occur in North Carolina (*337*), others have not found *Mycosphaerella rubi* Roark in the South-

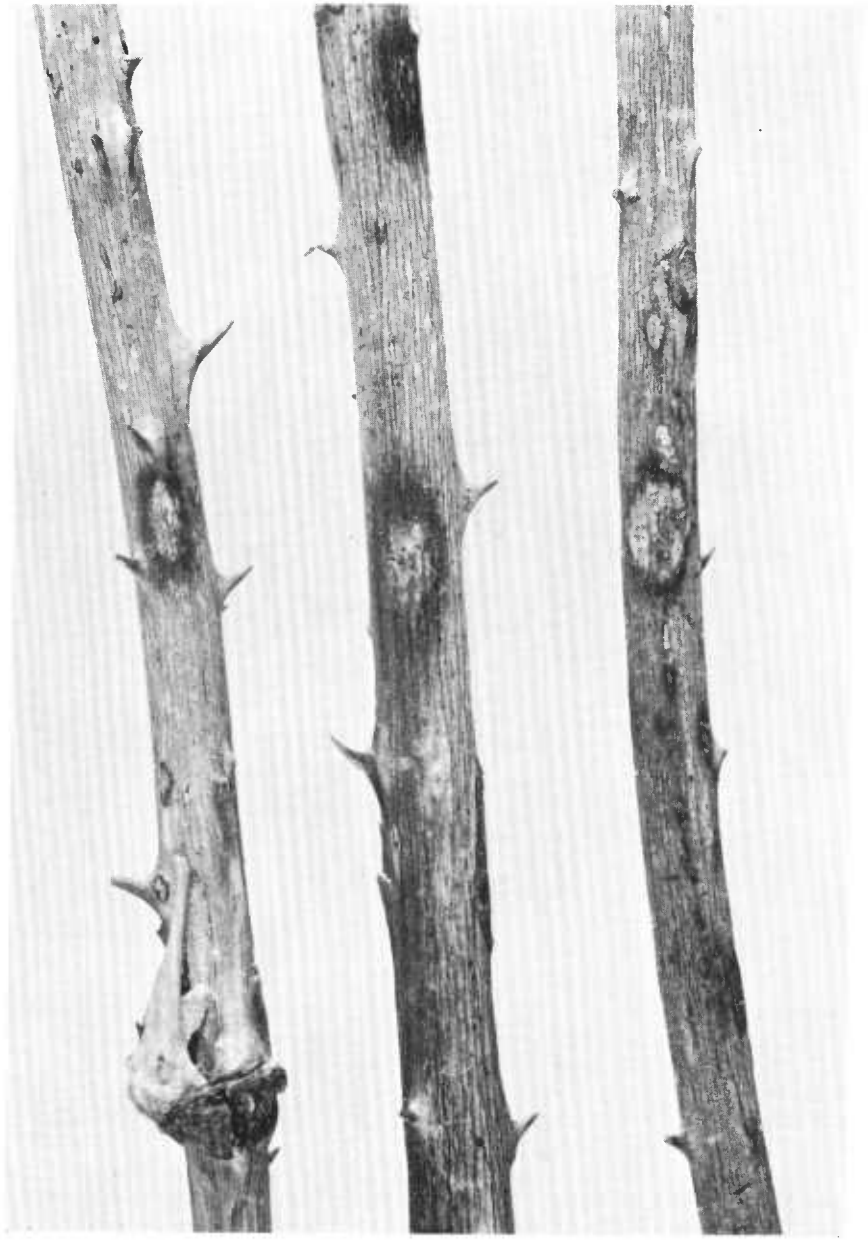

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FIGURE 41.—Septoria leaf spot disease on canes of trailing blackberry.

eastern States and conclude that the fungus overwinters in the pycnidial stage (*100*). Roark (*252*) did not successfully complete a cycle of controlled inoculations from ascospore to conidia and back to ascospore to establish relationship between *M. rubi* and *Septoria rubi* unequivocally.

Host resistance.—*Septoria rubi* is limited in host range to the genus *Rubus* and within that genus to certain species in the section *Eubatus* (*100*, *252*). The whole question of host resistance is related to the identity and distribution of the causal organism(s). In the Southeastern United States, the trailing blackberry varieties Lucretia, Youngberry, and Boysenberry are susceptible to leaf spot (*105*), while the trailing variety Carolina has some resistance (*43*). In the Pacific Northwest, trailing blackberry varieties such as Chehalem, Santiam, Youngberry, Loganberry, Boysenberry, derived from native western blackberries, and Lucretia are susceptible (*118*, *317*). Leaf spot resistance has been found in European blackberries in the Pacific Northwest (*317*). The Himalaya blackberry (*R. procerus* P. J. Muell.) is resistant to Septoria leaf spot, resistance being partially dominant and multifactorial (*284*).

The identity of the ascomycetous and related imperfect fungi causing the leaf spots of *Rubus* in the United States requires more study, to reconcile the conflicting reports in the literature as to the fungi involved and to confirm the existence of physiologic races, whose existence is already surmised (*100*, *252*) in one of the established fungus species of this group.

Control.—Pruning out and burning infected canes after harvest are desirable practices to lower the inoculum potential in the following spring (*118*). In the South, a delayed-dormant spray of lime-sulfur followed by three applications of ferbam or captan is the recommended schedule for leaf spot control on trailing blackberries (*105*). In the Pacific Northwest, a similar recommended three-spray schedule afforded some control on trailing blackberries but was not wholly satisfactory in years of severe leaf spot infection (*309*). Bordeaux mixture in September and a winter dormant application of lime-sulfur were added to the spray schedule in Oregon in years of severe leaf spot infection (*118*), but the dormant sprays were found to be ineffective in California (*332*).

Stamen Blight [33]

Zeller and Braun (*355*) described a stamen blight on wild and cultivated trailing blackberries in Oregon. The fungus was first described in Germany (*104*, *291*) and is of some importance in Scotland (*101*, *333*). Although minor, the disease has involved up to 30 percent of stamens, which do not dehisce but become covered with white fungal spore horns. The resulting poor pollination can interfere with fruit set when the disease is severe. Infections take place in the unopened fruiting buds.[34] A lime-sulfur spray in August has partly controlled the disease (*355*).

[33] Caused by *Haplosphaeria deformans* Syd.

[34] Blyth, W. Studies on *Haplosphaeria deformans* Sydow. 1949. [Unpublished doctor's thesis, 77 pp. Univ. Edinburgh.]

Haplosphaeria deformans is reported to be responsible for dry seedy fruits on several susceptible red rapsberries and blackberries in British Columbia (*68*), where the disease is known as anther and stigma blight. Varietal differences in susceptibility exist among blackberries and red raspberries. High humidity and poor air drainage favor the development of anther and stigma blight (*68*).

Verticillium Wilt [35]

Verticillium wilt of erect and trailing blackberries occurs in the Northern States and the Pacific Coast States. It may be serious on certain susceptible varieties. Verticillium wilt of blackberries was studied by Wilhelm and Thomas (*329*).

Symptoms.—Description of symptoms is drawn from California work with infected trailing blackberries (*329*). Young infected canes wilt; the leaves turn yellow and then become brown and necrotic. The advent of cool weather in the fall may bring about a virtual disappearance of symptoms. Canes do not turn blue as they do in the case of infected raspberry. Fruiting canes that survive the winter months leaf out and set fruit; but, as the fruit ripens in warmer weather, they usually collapse.

Casual organism and disease cycle.—See the discussions under raspberry, pages 47 to 49. Although data are not known to support the point, it is assumed that *Verticillium* isolates from all brambles will interinfect.

Host resistance.—Studies of resistance to Verticillium wilt in blackberries have been made on the Pacific coast (*271*, *330*, *352*). Many strains of *Verticillium albo-atrum* exist, differing in virulence; but good field resistance is known in many blackberries. Much of the resistance is derived from the wild California trailing blackberry (*Rubus ursinus* Cham. & Schlecht.) (*329*).

Resistant erect blackberry varieties include Evergreen, Himalaya, and Lawton; resistant trailing blackberries include Cascade, Chehalem, Loganberry, Olallie, and Phenomenal. *Rubus macropetalus* Dougl., the trailing blackberry of the Pacific Northwest, also is resistant. Boysenberry, Lucretia, Snyder, and Youngberry are susceptible.

Control.—Where practicable horticulturally, resistant varieties can be used. The control practices discussed for Verticillium wilt of raspberry (p. 51) are applicable to blackberry as well.

Bacterial Diseases

Cane Gall and Crown Gall

For a detailed description of cane gall, see page 54; a full description of crown gall is found on page 56.

Cane gall, reported by McKeen (*210*) and Coleman (*74*), occurs on blackberry in the Pacific Northwest.

Crown gall is widespread on blackberry (fig. 42). Although serious in nursery fields, where freedom from the disease is essential, crown

[35] Caused by *Verticillium albo-atrum* Reinke & Berth.

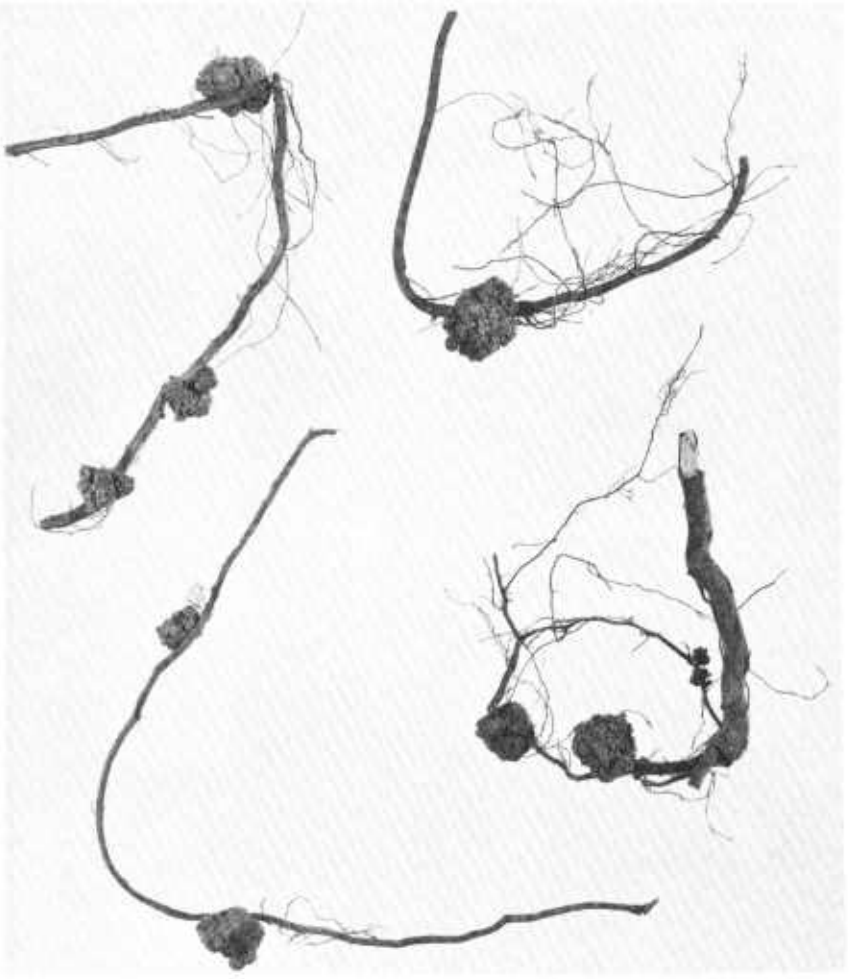

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FIGURE 42.—Crown gall of trailing blackberry.

gall is seldom a problem in fruiting fields, because blackberries grow so vigorously. Jones (*190*) studied the anatomy of crown gall growths on blackberry.

NEMATODES AND NONINFECTIOUS DISORDERS OF BRAMBLES

Nematodes

Little work has been done in North America on nematodes as parasites of *Rubus* roots or as vectors of *Rubus* viruses. An excellent text by Thorne (*298*) considers the biology of plant parasitic nematodes and the characteristics of the genera that have been found on *Rubus*.

Cadman (*57*) working in Scotland reviewed the nematode vectors (*Xiphinema* spp. and *Longidorus* spp.) of three viruses known in raspberry (see table 1).

Although no reports have as yet been published showing that North American nematodes move viruses into or out of *Rubus* species, *Xiphinema americanum* Cobb is known to be the vector of the peach yellow bud mosaic virus in peaches. This virus is a strain of the tomato ringspot virus. (See also p. 61). There is also evidence that raspberry ringspot, known to be nematode transmitted,[36] is caused by a strain of tomato ringspot virus (*280*). Peach yellow bud mosaic has been found to occur under field conditions in some *Rubus* cultivars in California (*193*).

Root lesion nematodes (*Pratylenchus* spp.) have been found in North America associated with roots of red or black raspberry (*27*, *68*, *138*, *139*, *173*, *303*), blackberry (*138*, *139*), and *Rubus* spp. (*35*). Often the plants are weakened when these nematodes are numerous. *Rubus* cultivars differ in their susceptibility to damage by *Pratylenchus* spp. (*139*). The role of root lesion nematodes in causing root damage in *Rubus* alone or in conjunction with other soil microorganisms may be quite important in *Rubus* root rot complexes and nonviral declines (*27*, *139*).

Some other nematodes found associated with *Rubus* roots in North America, sometimes in high populations and apparently causing direct crop losses, are: *Criconema* sp. (*35*, *68*) on raspberry and *Rubus* spp., *Criconemoides* sp. (*35*, *68*, *173*) on raspberry and blackberry, *Xiphinema americanum* Cobb (*35*, *68*, *173*) on raspberry and *Rubus* spp., and possibly *Hemicycliophora* sp. (*185*) on raspberry.

Although root-knot nematodes (*Meloidogyne* spp.) have been recorded on *Rubus*, Christie and Taylor (*71*) consider that raspberries and blackberries are in general resistant to these nematodes.

Morris (*219*) noted that late-maturing blackberry varieties were susceptible to damage by an unspecified nematode in Texas.

Noninfectious Disorders

Winter injury, poor soil drainage, and wind whipping all can seriously harm bramble plants. Often the symptoms produced are mistaken by growers for those of diseases caused by viruses or by fungi, and plants are needlessly removed. Likewise, some spray materials such as lime-sulfur and 2,4-dinitro-6- (2-octyl) phenyl crotonate can cause foliage mottling that resembles raspberry mosaic. Mineral deficiencies and excesses may produce symptoms, as in the chlorosis brought on by the lack of available iron in alkaline soils in the Western States (*305*). Many of the disorders connected with cultural practice are considered in books and articles dealing with the horticulture of the crops (*95*, *265*, *266*, *306*, *307*).

Winter Injury

Red raspberries are more liable to certain types of winter injury than black or purple raspberries or blackberries, particularly in the South-

[36] Stace-Smith, R. Personal communication, 1964.

eastern States, where growth occurs in the fall and repeated thawing and freezing occur in the spring. Red raspberry canes or whole plants that are not sufficiently hardened in the fall may be killed by low winter temperatures. Slight killing of cane tips is common, but this is not of much importance since cane tips normally are pruned off in the spring anyway. Often winter injury to canes occurs at cane bases, and the effects (wilting and dying) may not be evident until summer. Also, canes damaged by cold weather will have brownish wood under the bark later in the growing season instead of the normal greenish wood (*266*), although this symptom may also develop with certain root rots. The Latham red raspberry is notable for its ability to withstand winter injury. These plants when severely winter injured in one season can recover and produce well the next. Buds that begin to swell in spring thaws may later be killed outright or weakened.

Direct cold damage to all bramble crops can result in death of plants, or severe weakening. This subject is well reviewed by Shoemaker (*265*).

Light frost damage to raspberry buds may result in the production of mottled or stippled leaves that may be mistaken for mosaic-infected or mite-infested leaves (*25*). Frost stippling is usually confined to faint streaks paralleling veins. Such frost damage does not harm raspberries, but more severe frosts may blight flowers and young shoots.

Control.—To avoid injury from winter cold, growers should maintain vigorous growth of bramble crops in the spring and summer; but in the fall, so as to discourage growth, they should not fertilize or cultivate. In very cold areas, to protect them over the winter, canes are arranged on the ground and covered with dirt and in the following spring are trained up again (*265*). In areas with milder winters, growers sometimes leave old canes in the field over the winter to reduce wind damage to new canes. The practice is dangerous because so many of the *Rubus* fungi live over on dead canes. Control of defoliating pests in late summer helps to maintain vigor and lessen winter damage.

Wind Damage

Young black raspberry canes have a very weak point of attachment to the crown and are easily broken by wind at this point (*25*). Wilting and death of such wind-damaged canes is very common in the spring, and the canes may be mistaken for those infected with *Verticillium* (see p. 46). The wind-damaged canes, unlike the infected ones, can be easily pulled up. Exposed planting sites should be avoided where wind is a problem. Supporting black and purple raspberries on posts or wire and heading plants low reduces wind damage (*266*). It is often desirable to lessen damage from winter winds by planting a cover crop in August between the rows of a bramble crop (*266*). Cultivation too close to crowns will likewise cause new canes to break.

Poor Soil Drainage

Raspberries and blackberries are very intolerant of poorly drained soil, particularly in the wintertime (*25*). In wet soils roots and crowns are weakened and become subject to attack by soil microorganisms (p. 41). In choosing planting sites, deep well-drained soil not overlying a hardpan should be sought. Drainage and tiling permit good raspberry crops to be grown on otherwise unsuitable soils (*248*).

Fasciation

Occasionally unusually large fasciated primocanes will develop in black raspberries. Fulton (*133*) noted and illustrated them and found that plants fasciated one season were normal the next year. The reasons for this fasciation are unknown.

Crumbly Berries

A condition of unknown etiology is common wherever red raspberries are grown, wherein the individual drupelets of the fruits do not cohere well together and fall apart when picked (fig. 43). Certain varieties such as Latham and Indian Summer (*266*) and Washington (*68*) are particularly susceptible.

Normal and crumbly clones of a red raspberry selection were compared by the author. The average number of unfertilized pistils per fruit from the normal clone was 0.8, compared with 20.4 per fruit from the crumbly clone. Individual crumbly fruits weighed 41 percent of normal fruits.

Various possible causes for crumbliness have been suggested. They include: Genetic or chromosomal abnormalities (*159*, *187*), mosaic viruses (*78*, *266*, *303*), mild streak virus (*182*), unknown viruses (*159*, *248*, *303*), winter injury (*159*, *182*, *248*, *266*, *303*), mineral deficiencies (*159*, *182*, *303*), water relations (*303*), and insect injury (*159*). Several of these factors working together are suggested by most workers to produce the crumbly condition. A thoroughgoing study of the problem has yet to appear.

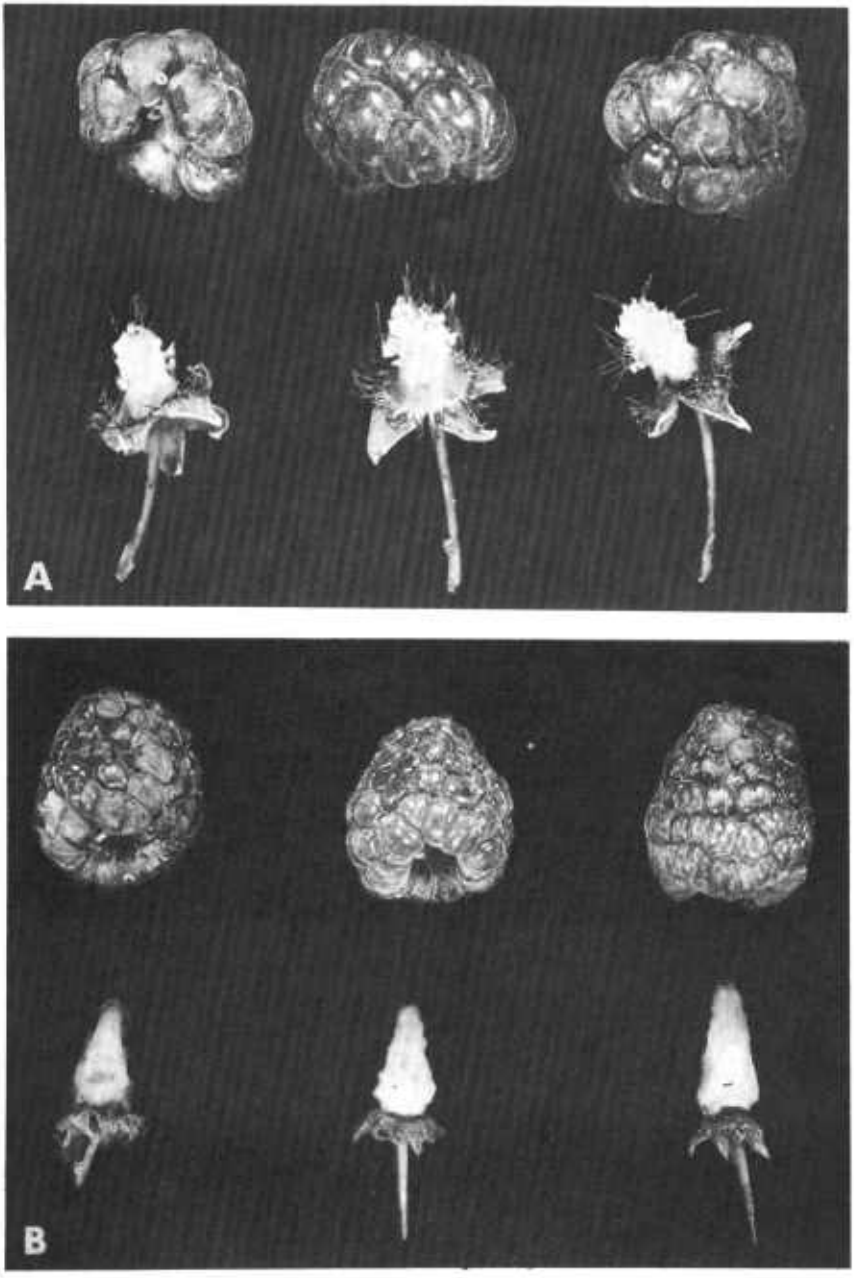

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FIGURE 43.—*A*, Crumbly red raspberries. Note the persistent aborted pistils on the receptacles from which these fruits have been detached. *B*, Normal berries of the same selection with their receptacles virtually free of aborted pistils.

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APPENDIX

Key to Major Diseases of Cultivated Brambles in the United States

This key is based on field symptoms and signs of the more important bramble diseases. These symptoms and signs may vary with the variety, the area, and the season. Preliminary diagnosis can be made by comparing symptomatology with the descriptions listed in this handbook or in the pertinent literature citations. Conclusive diagnosis of fungus and bacterial diseases, and determination of the organisms found associated with them, should be based on laboratory examination. Critical work, of course, requires the establishment of the pathogenicity of an isolate of the suspected pathogen.

For virus diseases where symptoms are not diagnostic, identification can be made by sap, graft, or vector transmissions to special indicator hosts; in some cases, serological tests can be made.

Best use of the Key requires that the host bramble or brambles be known; and some assumption, based on judgment, must be made about the nature of the causal agent of the disease—whether fungus, bacterium, or virus. In general, the spore-bearing structures of pathogenic fungi can be seen in or on the host, particularly with the aid of a hand lens. Usually such fungus structures develop in lesions. In the Key, miscellaneous root-rotting fungi and nematodes are grouped together in three places, according to the type of bramble host. Necrotic roots infected by fungi and nematodes require microscopic examination for identification of causal agent. Bacterial diseases of brambles cause either galls or necrotic shoots without discrete lesions. Virus diseases are the most difficult to diagnose. Many virus diseases are latent in brambles, causing only a generalized reduced vigor. Such viruses must be detected indirectly by indexing methods. Many abnormalities of the bramble plants are due to environmental causes, such as temperature extremes, high winds, or nutritional imbalances, or, to insect damage. With only a few exceptions, these are not included in this Key.

The Key is dichotomous, and each disease listed is followed by the number of the page in the handbook where it is described and in many cases illustrated.

The Key is divided into seven main sections: I. Virus diseases of black and purple raspberries; II. Virus diseases of red raspberries; III. Virus diseases of blackberries; IV. Fungus diseases of black and purple raspberries; V. Fungus diseases of red raspberries; VI. Fungus diseases of blackberries; and VII. Bacterial diseases of brambles.

I. Virus diseases of black and purple raspberries.

A_1 Leaves curled.

B_1 Leaves small, in rosettes, deeply ribbed; plants dwarfed; berries small, dry and seedy ..... *Leaf curl*, p. 9

B_2 Leaves of normal size, not ribbed; tips of leaflets hooked or rolled under; fruit juicy, lacking gloss and flavor.

C_1 Leaves often rugose and faintly mottled, or having yellow veins; normally spaced on vigorous canes; primocanes with faint blue streaks at bases ..... *Mild streak*, p. 5

C_2 Leaves often in rosettes and distinctly mottled; primocanes strongly streaked; plants weak ..... *Severe streak*, p. 3

A_2 Leaves not curled.

B_1 Young foliage often mottled and blistered or with cleared veins; shoot tips sometimes dead and blackened ..... *Raspberry mosaic*, p. 11

B_2 No viruslike symptoms; plants often weak; virus detection in indicator plants or by serological means ..... *Raspberry mosaic*, p. 11 *Raspberry bushy dwarf*, p. 19 and *Miscellaneous viruses*, p. 18

II. Virus diseases of red raspberries.
- A_1 Leaves strongly curled; in rosettes and deeply ribbed; plants severely dwarfed; berries small, dry, and seedy ..... *Leaf curl*, p. 9
- A_2 Leaves not strongly curled or ribbed.
  - B_1 Virus symptoms present.
    - C_1 Young leaves mottled or veins cleared ..... *Raspberry mosaic*, p. 11
    - C_2 Young leaves with transient ring spots ..... *Raspberry ringspot*, p. 19
  - B_2 Virus symptoms absent; plants often weak; virus detection in indicator plants ..... *Raspberry mosaic*, p. 11; *Raspberry bushy dwarf*, p. 19; and *Miscellaneous viruses*, p. 18

III. Virus diseases of blackberries.
- A_1 Limited to Loganberry.
  - B_1 Leaves with brilliant yellow blotches ..... *Calico*, p. 62
  - B_2 Pale green leaves with mottle; older leaves reddened and often cupped downward; plants dwarfed; rosettes and witches'-brooms often present ..... *Dwarf*, p. 62
- A_2 Not limited to Loganberry.
  - B_1 Viruslike symptoms on leaves.
    - C_1 Chlorotic spots, oakleaf patterns, or vein clearing *Blackberry mosaic*, p. 60
    - C_2 Not as above.
      - D_1 Somewhat angular white blotches ..... *Variegation*, p. 64
      - D_2 Mottling ..... *Raspberry mosaic*, p. 60
  - B_2 No viruslike symptoms on leaves.
    - C_1 Plants normal sized; all or most drupelets aborted ..... *Sterility*, p. 63
    - C_2 Plants small and weak; no drupelets aborted; virus detection in indicator plants ..... *Raspberry mosaic*, p. 60; and *Miscellaneous viruses*, p. 65

IV. Fungus diseases of black and purple raspberries.
- A_1 Leaves with lesions.
  - B_1 Lesions with light-colored centers and purple edges.
    - C_1 Lesions irregular in shape, small, with light gray centers, later often dropping out, often coalesced; cane lesions light gray with purple edges, coalesced into cankers which split open to give a rough, knobby appearance; fruit often dry and shriveled *Anthracnose*, p. 21
    - C_2 Lesions circular, usually dotted with small black fruiting bodies; severely infected leaves shed; cane lesions inconspicuous near cane bases ..... *Leaf spot*, p. 34
  - B_2 Pustules of orange spores on lower surfaces of young leaves; infected canes lacking spines, often numerous and willowy *Orange rust*, p. 36
- A_2 Leaves without lesions.
  - B_1 Leaves whitish or prematurely yellow.
    - C_1 Leaves with diffuse, whitish blotches, often mottled, water-soaked on lower side; no death of canes ..... *Powdery mildew*, p. 39
    - C_2 Leaves prematurely yellow; many canes dead.
      - D_1 Plants killed in circular areas of the field; thick sheets of white mycelium under the bark of roots. Rare except in Southwest United States ..... *Armillaria root rot*, p. 42
      - D_2 Plants not killed in circular areas of the field; no white mycelium under bark of dying canes.
        - E_1 Leaves on fruiting canes turn yellow and drop progressively from base upward; infected primocanes wilt, turn blue and die; canes develop no cankers; roots and cane bases often have dark-colored wood ..... *Verticillium wilt*, p. 46
        - E_2 Leaves on fruiting laterals become yellow and die near brown, fissured cane cankers; pith of infected canes brown and crumbly; black fruiting bodies formed in epidermis of infected canes, producing a gray smoky appearance ..... *Cane blight*, p. 26

B_2 Leaves symptomless.
C_1 Plants weak; fruits normal; roots necrotic---- *Miscellaneous root rotting fungi*, p. 41 and *Nematodes*, p. 83
C_2 Fruits rotted.
D_1 Mycelium visible on fruit.
E_1 Mycelium covering single drupelets or entire fruits before harvest or in storage, forming dark dusty spore masses *Gray mold*, p. 29
E_2 Mycelium covering entire fruits, in storage only.
F_1 Mycelium white, turning blue green---- *Blue mold rot*, p. 29
F_2 Mycelium dark.
G_1 Watery fruit rot covered with coarse gray mycelium bearing black aerial fruiting bodies-- *Rhizopus rot*, p. 31
G_2 Firm fruit rot covered with gray mycelium *Alternaria rot*, p. 29
D_2 Mycelium not visible on fruit.
E_1 Immature berries dry and hard; no characteristic lesions *Brown berry*, p. 31
E_2 Fruit rotting in storage, olive-green mycelium inside the berry cup---------- *Cladosporium rot*, p. 29

V. Fungus diseases of red raspberries.
A_1 Leaves with lesions.
B_1 Lesions with light-colored centers and purple edges.
C_1 Lesions irregular in shape, with small, light-gray centers; often coalesced; cane lesions small, grayish, sunken or raised, or rings of black fruiting bodies on unbroken epidermis *Anthracnose*, p. 21
C_2 Lesions circluar, usually dotted with small black fruiting bodies; severely infected leaves shed; cane lesions inconspicuous near cane bases---------- *Leaf spot*, p. 34
B_2 Lesions without light-colored centers and/or purple edges.
C_1 Pustules of orange or yellow spores.
D_1 Pustules of powdery yellow spores on undersides of mature leaves; no cane cankers---------- *Late leaf rust*, p. 32
D_2 Small, scattered, orange-yellow pustules on upper surfaes of young leaves; pale yellow pustules on lower surfaces of leaves after fruiting; cane cankers---------- *Yellow rust*, p. 52
C_2 Lesions large, brown, with yellow edges, wedge shaped; infected nodes brown; buds shrivel and die--------- *Spur blight*, p. 42
A_2 Leaves without lesions.
B_1 Leaves whitish or prematurely yellow.
C_1 Leaves with diffuse whitish blotches, often mottled; water-soaked on lower side; canes may be spindly but do not die *Powdery mildew*, p. 39
C_2 Leaves prematurely yellow; many canes dead.
D_1 Cane cankers on overwintering canes.
E_1 Brown, fissured cankers; pith of infected canes brown and crumbly; black fruiting bodies formed in epidermis of infected canes, producing a gray smoky appearance *Cane blight*, p. 26
E_2 Infected canes with distinctive "watermark" symptoms showing through epidermis---------- *Gray mold*, p. 29
D_2 No cane cankers.
E_1 Leaves on fruiting canes turn yellow and drop progressively from base upward, often leaving a tuft of leaves at the tip; primocanes may wilt and occasionally turn blue and die; roots and cane bases often have dark-colored wood *Verticillium wilt*, p. 46
E_2 Plants killed in circular areas of the field; thick sheets of white mycelium under the bark of roots. Rare except in Southwest United States---------- *Armillaria root rot*, p. 42

V. Fungus diseases of red raspberries—Continued
  A_2 Leaves without lesions—Continued
    B_2 Leaves symptomless.
      C_1 Plants weak, roots necrotic—*Miscellaneous root rotting fungi*, p. 41 and *Nematodes*, p. 83
      C_2 Flowers or fruits infected.
        D_1 Flowers infected; anthers covered with white fungus growth; stigmas sometimes covered also—*Stamen blight*, p. 81
        D_2 Flowers normal; fruit infected.
          E_1 Mycelium visible on fruit.
            F_1 Mycelium covering single drupelets or entire fruits before harvest or in storage, forming dark dusty spore masses—*Gray mold*, p. 29
            F_2 Mycelium covering entire fruit in storage only.
              G_1 Mycelium white, turning blue green—*Blue mold rot*, p. 29
              G_2 Mycelium dark.
                H_1 Watery fruit rot covered with coarse gray mycelium bearing black aerial fruiting bodies—*Rhizopus rot*, p. 31
                H_2 Firm fruit rot covered with dark gray mycelium—*Alternaria rot*, p. 29
          E_2 Mycelium not visible on fruit; fruit rotting in storage, olive-green mycelium inside the berry cup—*Cladosporium rot*, p. 29

VI. Fungus diseases of blackberries.
  A_1 Leaves with lesions.
    B_1 Pustules of yellow or orange spore masses on undersides of leaves.
      C_1 Pustules containing orange waxy spores; witches'-brooms often present—*Orange rust*, p. 72
      C_2 Pustules containing lemon-yellow powdery spores on canes as well as leaves. No witches'-brooms—*Cane and leaf rust*, p. 68
    B_2 Lesions with no rust spore masses present.
      C_1 Lesions with light-colored centers and purple edges.
        D_1 Numerous, small, black fruiting bodies in centers of moderately large lesions; also on canes—*Leaf spot*, p. 78
        D_2 No such fruiting bodies; lesions irregular in shape, small, with light-gray centers, often coalesced; cane lesions small, grayish, and sunken—*Anthracnose*, p. 66
      C_2 Large, brown, irregularly shaped leaf spots. Occurring especially in Southeast United States—*Blotch*, p. 79
  A_2 Leaves without lesions.
    B_1 Leaves prematurely yellow; many canes dead.
      C_1 Cankers on fruiting canes containing small black fruiting bodies—*Cane blight*, p. 68
      C_2 Bark not cankered.
        D_1 Plants wilted or killed in circular areas of the field.
          E_1 White mycelium on surface of roots, disease spreads slowly. Primarily on trailing blackberries in Southeast United States—*Collybia root rot*, p. 42
          E_2 Thick sheets of white mycelium under the bark of roots. Disease spreads rapidly. Rare except in Southwest United States—*Armillaria root rot*, p. 42
        D_2 Plants not necessarily killed or dying in circular areas of the field; white mycelium not formed in or on roots.
          E_1 Leaves wilted, turning progressively yellow and brown; roots and cane bases often have dark-colored wood—*Verticillium wilt*, p. 82
          E_2 Plants weak, not usually wilted; wood of cane bases not typically discolored; roots are necrotic to varying degrees—*Miscellaneous root rotting fungi*, p. 41 and *Nematodes*, p. 83
    B_2 Leaves not prematurely yellowing; canes rarely dead.
      C_1 Symptoms principally on canes.
        D_1 Witches'-brooms common; petals pink, twisted, and enlarged—*Rosette*, p. 76

D_2 No witches'-brooms formed.
E_1 Buds dead on overwintering canes. Black mustard-seed-like sclerotia in infected areas ..... *Gray mold*, p. 71
E_2 Buds not dead; large brown patches on overwintering canes ..... *Cane spot*, p. 78
C_2 Symptoms principally on fruit and flowers.
D_1 Flowers infected, anthers covered with white fungus growth; fruit may be malformed and infected drupelets covered with small, black fruiting bodies ..... *Stamen blight*, p. 81
D_2 Flowers normal; fruit infected.
E_1 Mycelium visible on fruit.
F_1 Watery fruit rot in storage, or berries mummified in the field.
G_1 Fine, dark-gray mycelium with dusty masses of dark spores covering fruit.
G_2 Coarse, light-gray mycellium with black aerial fruiting bodies ..... *Rhizopus rot*, p. 31
F_2 Fruit firm, not collapsing in storage or becoming mummified in the field.
G_1 Mycelium white, turning blue green ..... *Blue mold rot*, p. 29
G_2 Mycelium dark.
H_1 Small black fruiting bodies slowly developing on scabby drupelets ..... *Black rot*, p. 71
H_2 No such fruiting bodies.
I_1 Mycelium dark gray, covering berries *Alternaria rot*, p. 29
I_2 Mycelium sparse, olive green ..... *Cladosporium rot*, p. 29
E_2 Mycelium not visible on fruit.
F_1 Berries dry, shriveled and crumbly ..... *Dry berry*, p. 71
F_2 Berries not dry, shriveled or crumbly, variously colored, often with white drupelets ..... *Redberry*, p. 71

VII. Bacterial diseases of brambles.
A_1 Galls or warts present.
B_1 On canes above ground (red raspberries not susceptible) ..... *Cane gall*, p. 54
B_2 On crown and roots, at and below ground line ..... *Crown gall*, p. 56
A_2 No galls or warts; shoot tips, leaves, or flowers turn black and die ..... *Fire blight*, p. 59

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Caution: Use pesticides only when needed and handle them with care. If pesticides are handled or applied improperly, or if unused parts are disposed of improperly, they may be injurious to humans, domestic animals, desirable plants, pollinating insects, fish or other wildlife, and may contaminate water supplies. Follow the directions and heed all precautions on the container label.